

Pathogenesis of periodontal diseases

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2016

Clinically healthy gingiva



Classical experimental gingivitis study by Loe



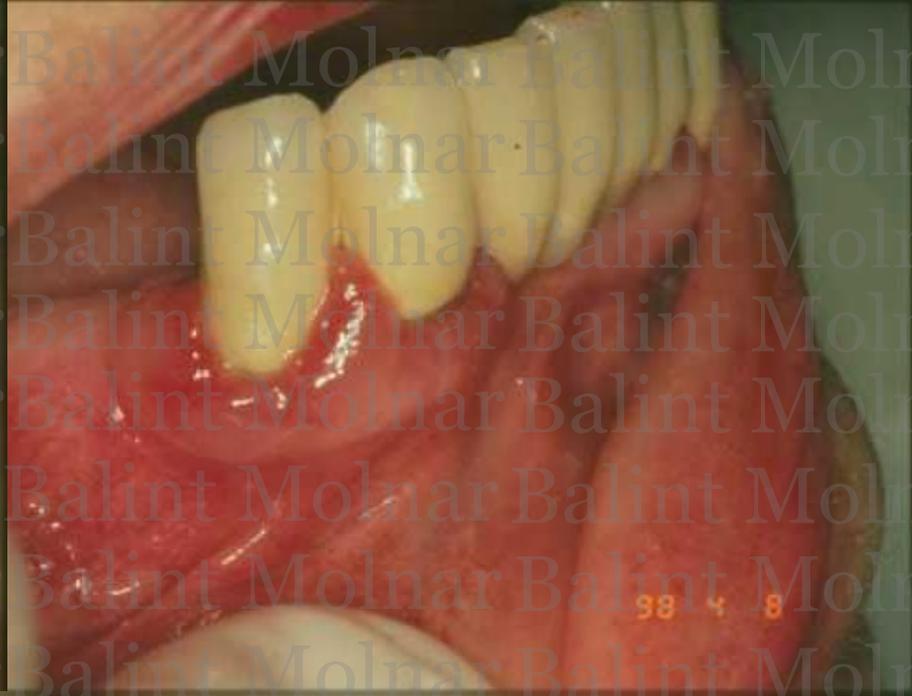
DENTAL PLAQUE



GINGIVITIS



PERIODONTITIS



Gingivitis



Periodontitis

Irreversible damage of the attachment apparatus

Insufficient immune response

Periodontal immune reactions

- ▶ Local immunoglobulin secretion
- ▶ Phagocytosis by PMN cells in the sulcus
- ▶ Complement system
- ▶ Intact epithel barrier
- ▶ Wetting and washing effect of the crevicular fluid



Primary and secondary immune response

► Primary immune response

- Non-specific, immediately activated, doesn't increase over time
- No memory cells, no adaptation

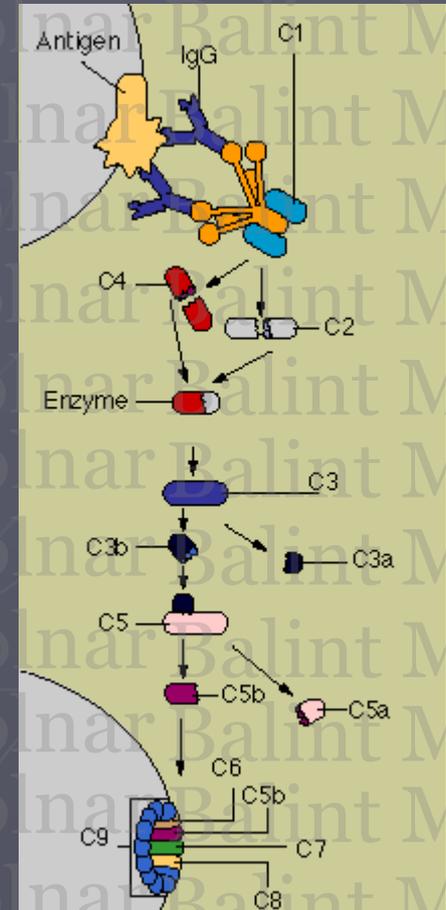
► Secondary immune response

- Specific immune response, followed by activation, increases over time
- Adaptation of memory cells

Primary immune response

Humoral elements I

- ▶ Protheolytic enzymes
 - Mainly endopeptidases
 - MMPs, collagenases
- ▶ Complement system
 - Membrane receptors and soluble glycoproteins
 - C3a C5a stimulate mast cells
 - C3b (Opsonin) induces opsonisation

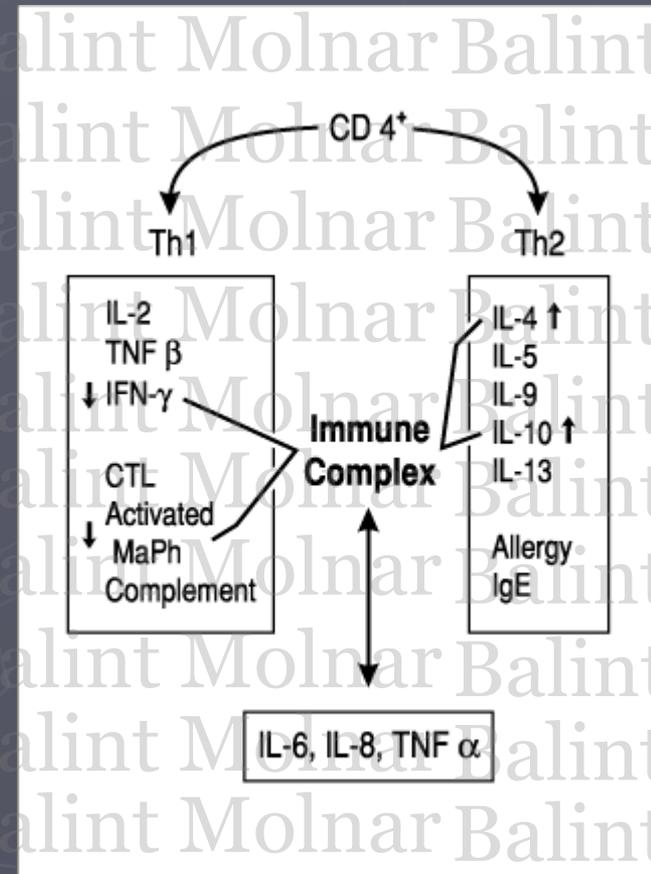


Primary immune response

Humoral elements II

► Cytokines

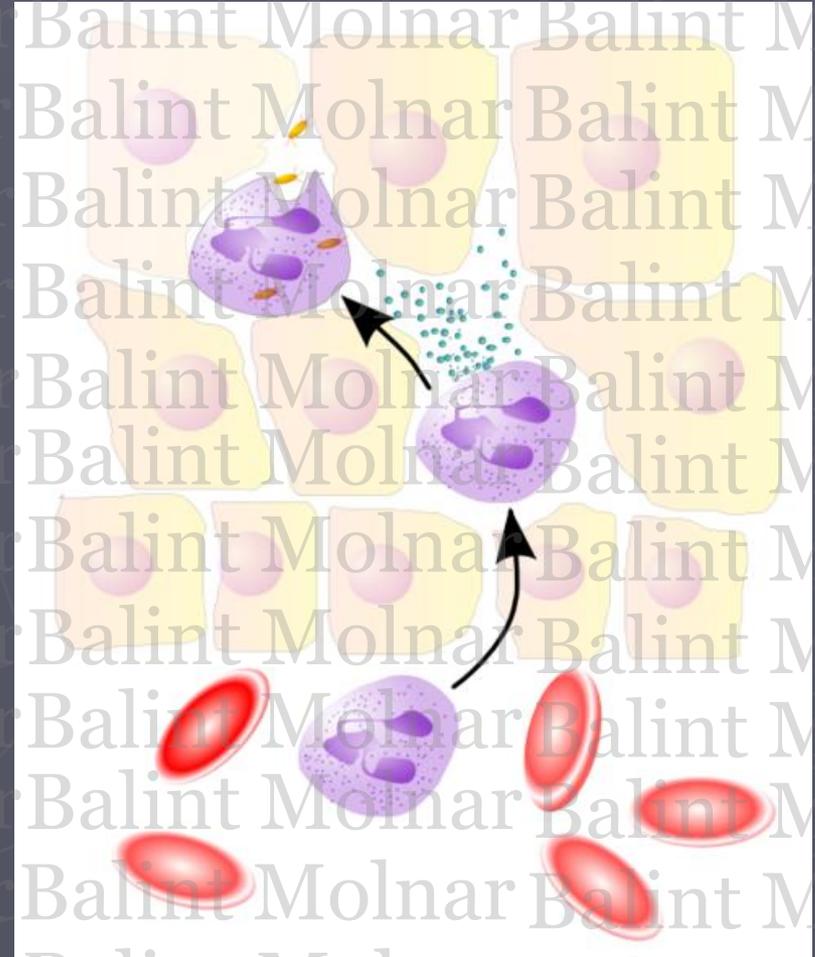
- Small proteins, acting in intercellular communication
- Soluble molecules with paracrin and autocrin effects
- Inflammatory cytokines
citokinek: IL-1, IL-6, TNF α , IFN β , γ
- Chemotactic cytokine: IL-8
- Arachidonic acid metabolites: Prostaglandin, Leukotrien



Primary immune response

Cellular elements I

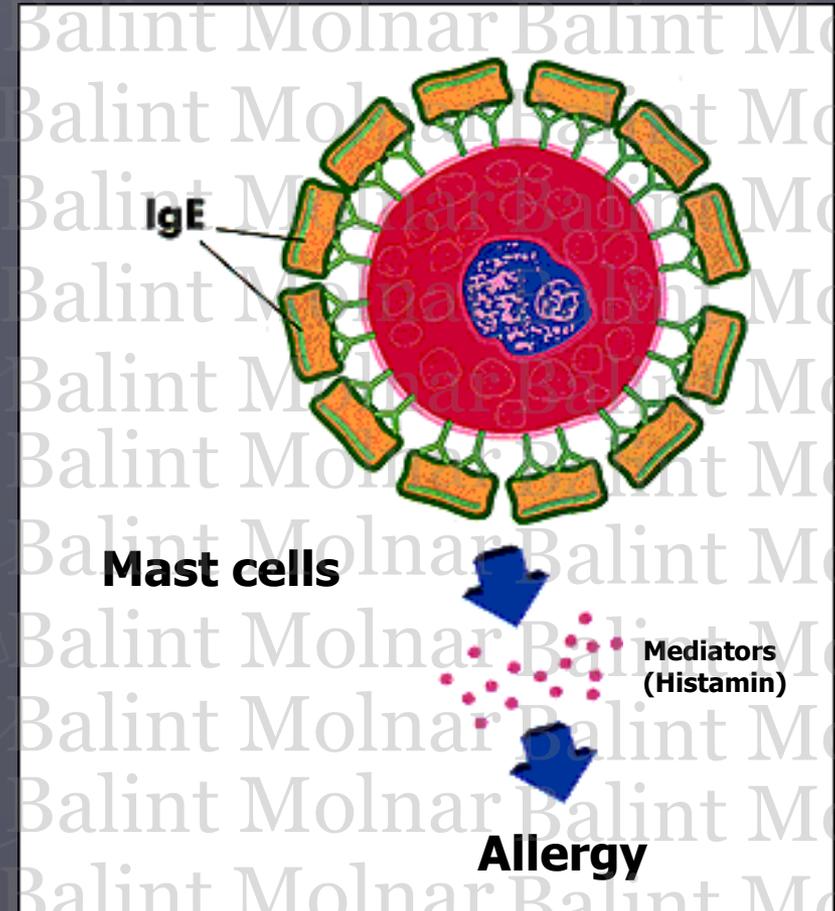
- ▶ Neutrophil granulocytes
 - Majority of WBC
 - Numerous granules
 - Functions:
 - Chemotaxis,
 - Diapedesis,
 - Migratio,
 - Phagocytosis,
 - Opsonisatio



Primary immune response

Cellular elements II

- ▶ Basophylil granulocytes and mast cells
 - 1-2% of WBC
 - Granules: Histamine, Leukotriene, Heparine and Serotonin
 - Regulate most important inflammatory processes (Vasodilatation and oedema) and anaphylaxis
- ▶ Mononuclear antibodies
 - ▶ Langerhans-cells (Antigen presentation)
 - ▶ Macrophags (phagocytosis)

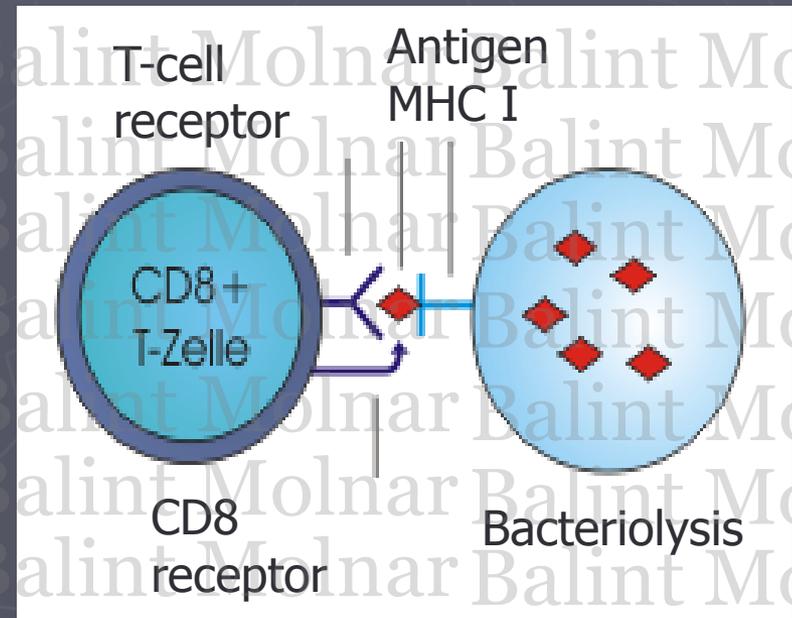


Secondary immune response

Cellular elements I

► T-Lymphocytes

- TCR (T-cell receptors)
- Three subgroups: cytotoxic T-cells (CD8+), helper cells (CD4+), regulatory T-cells (CD4+)
- MHC I activation, fight against bacteria, first specific reactions in periodontitis

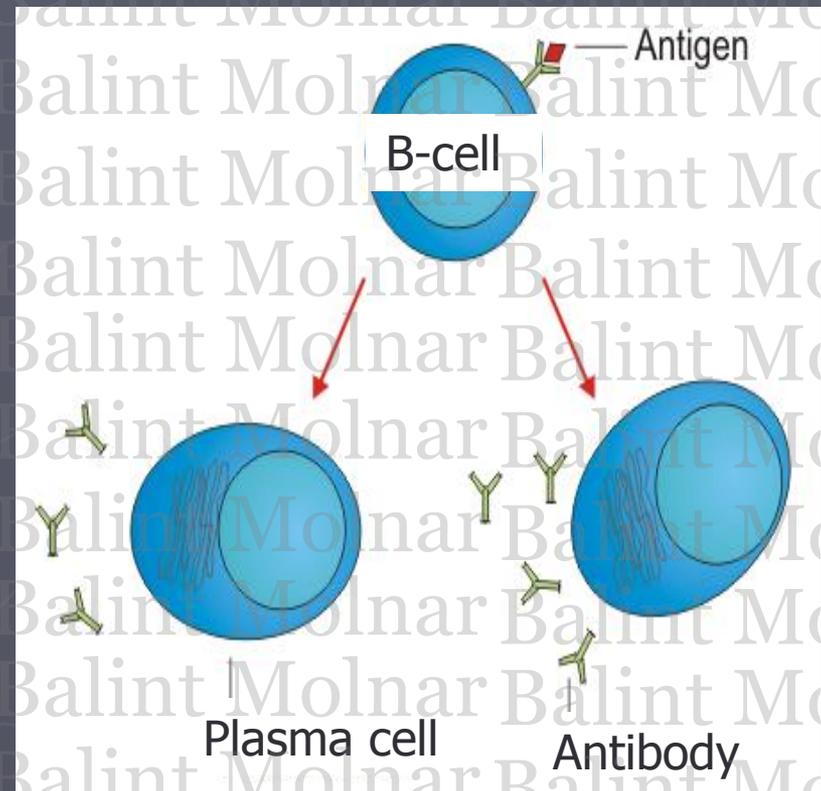


Secondary immune response

Cellular elements II

► B-Lymphocytes

- BCR (B-Cell receptors), soluble antigen detection
- Antigen presentation via MHC II
- Activated plas cells: antibody secretion
- 10-15% of lymphocytes

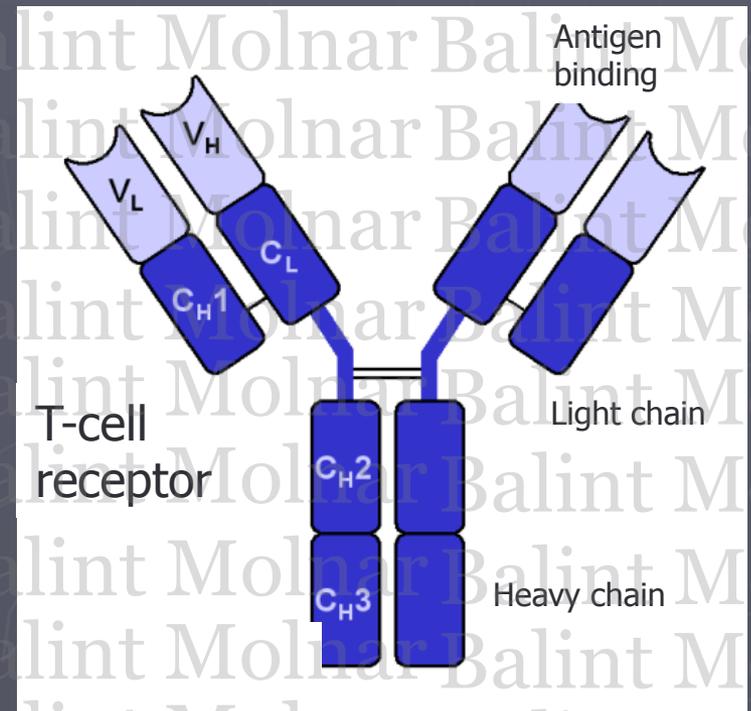


Secondary immune response

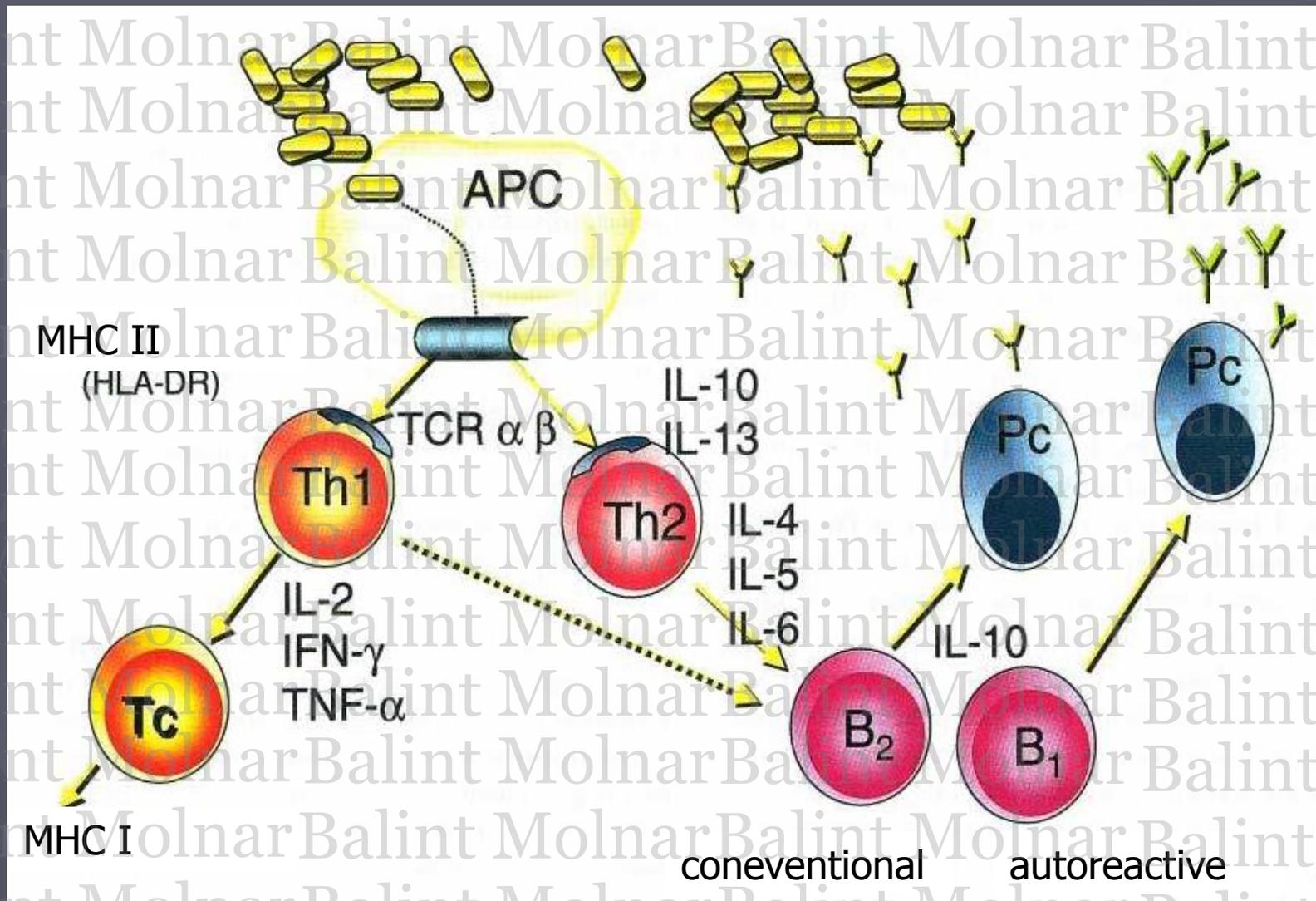
Humoral elements

► Antibodies (Immunglobulines)

- IgM is most important (Opsonisation, fixation of bacteria)
- IgG: more subclasses, IgG3 most effective
- IgA is the first defense line at the mucosa, inhibits bacterial adhesion

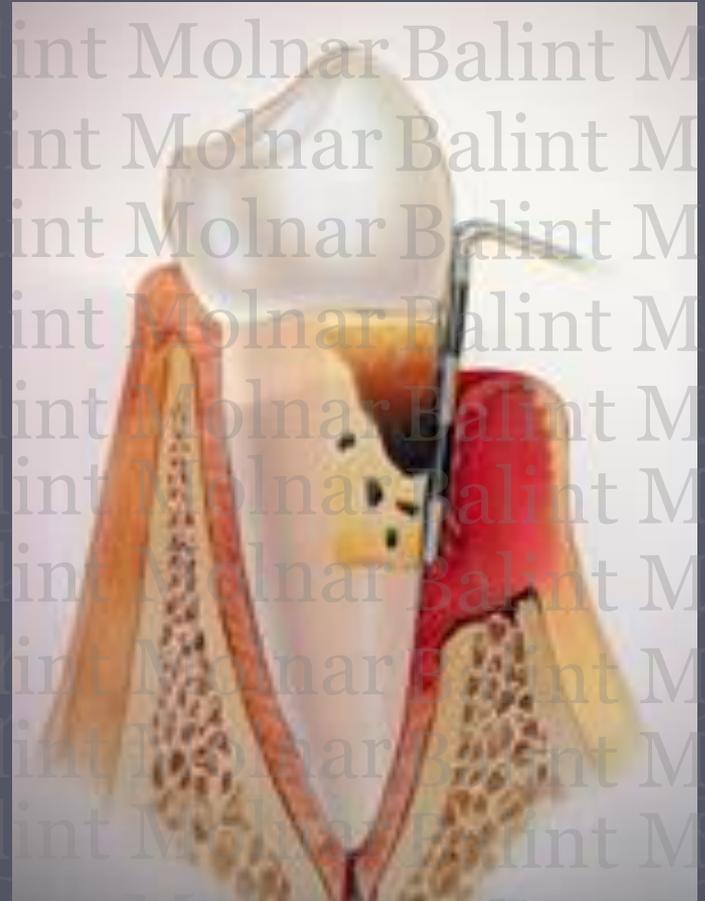


Immune response in periodontitis



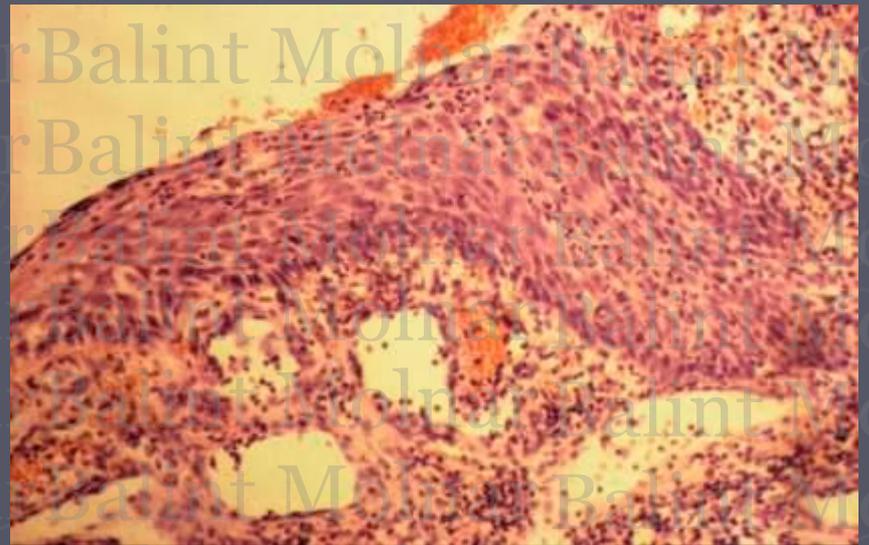
Mechanisms of periodontal tissue breakdown

- ▶ Direct bacterial factors
 - Soluble enzymes
 - Ammonium and Indol secretion
 - LPS
- ▶ Role of host immune response
 - Related to bacterial antigenes, endo- and exotoxines tissue damaging reactions are initiated



Histology

Damaged tissues are filled with gingival capillary vessels and cellular elements



Mechanisms of connective tissue loss

Matrix Metalloproteinases

MMP secretion: PMN cells, macrophages, epithelial cells, fibroblasts

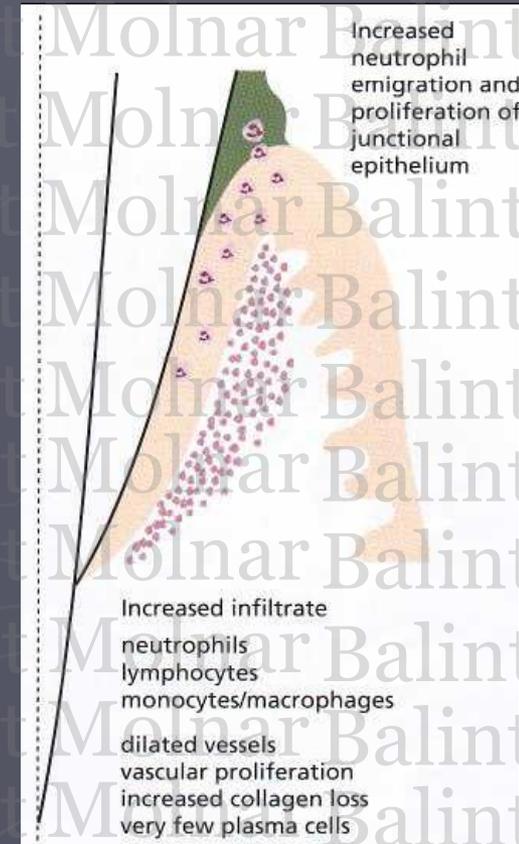
MMP8: PMN cells, MMP1: macrophages

Protection of gingival metalloproteines: Doxycycline

Clinical and histological observations I

► Gingivitis:

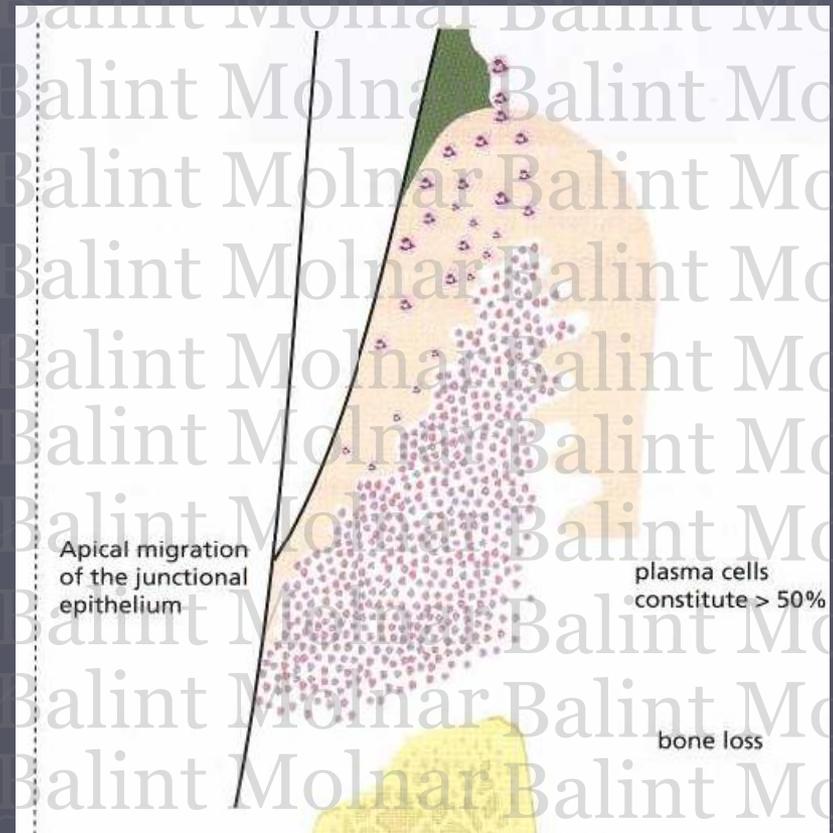
- Clinically: Vasodilatatio, Erythema, Oedema, bleeding
- Histology: cellular content of soft tissues 10-15%, mainly PMN cells, T.Lymphocytes, Macrophages
- Capillary proliferation, tissue damage



Clinical and histological observations II

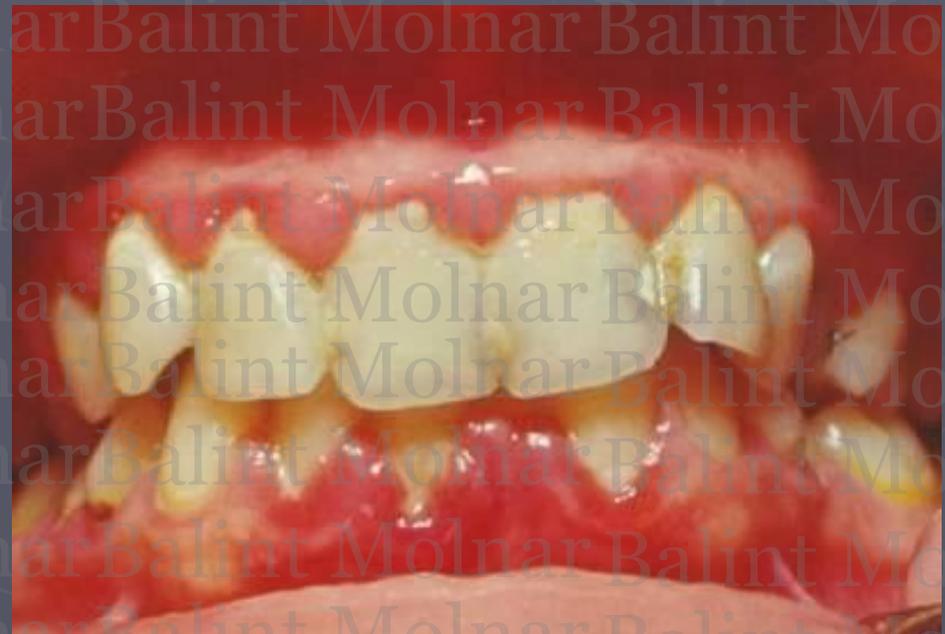
► Periodontitis:

- Clinically: Vasodilatatio, Erythema, Oedema, bleeding, attachment loss!
- Histology: cells content of soft tissues over 60%, mainly T- B- Lymphocytes, Macrophages, Plasma cells (over 50%)
- Capillary proliferation, collagen degradation
- Fibroblast degeneration, pocket formation, bone loss



Immunological background I

- ▶ **Chronic Gingivitis**
 - Strong primary immune response
 - Cellular immunoreactions
 - Protective antibody secretion
 - Stabil, no progression



Immunological background II

► Destructive Periodontitis

- Weak primary immune response
- Aggressive periodontopathogenic bacteria
- Polyclonal B-cell stimulation, Th2 response
- No protective antibody secretion
- Progressive lesion



Thank you for your attention!

