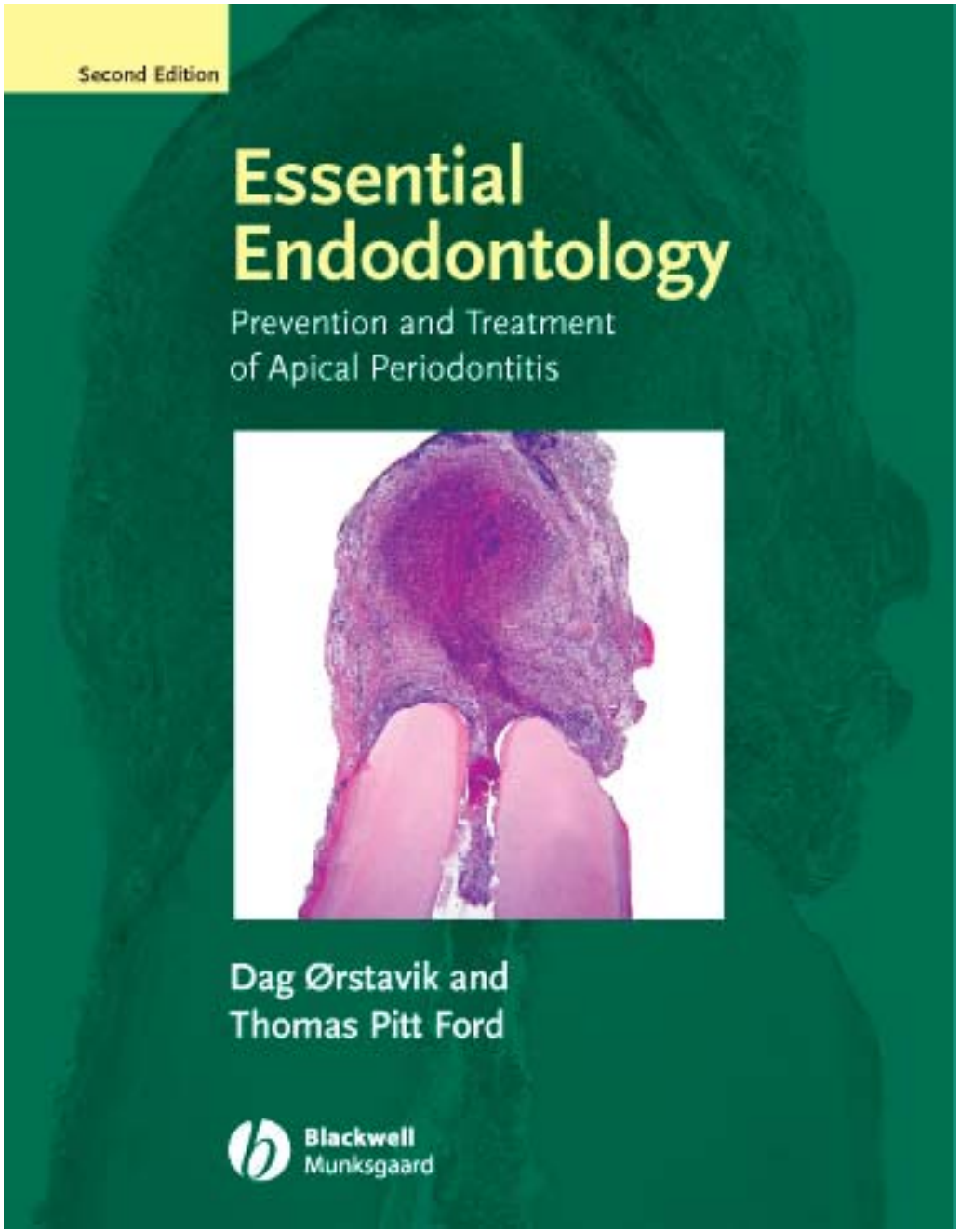

Endodontic Topics 2002 -

Visual Endodontics

A skull of a woman from a heathen grave at Hólaskógi in Thjórsárdal. It is probable that a dental infection in the upper jaw was the cause of her death.

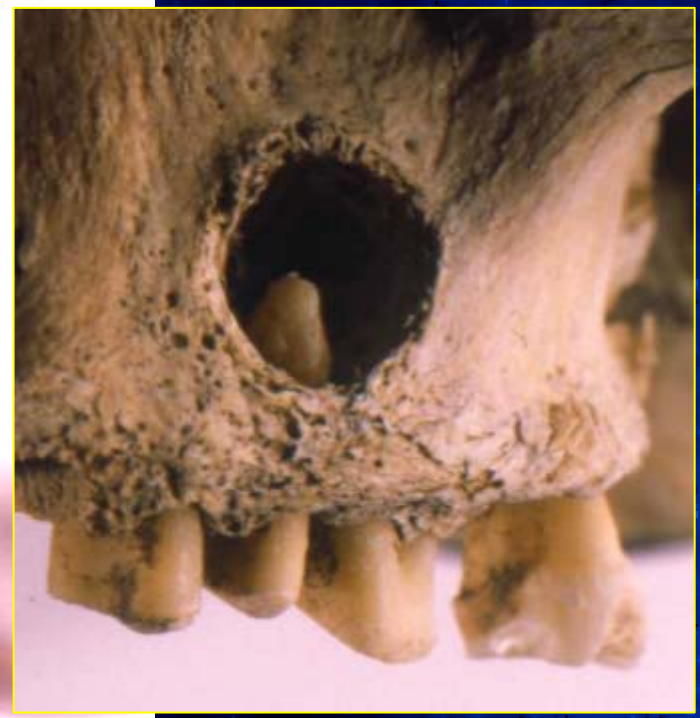

Photo and text of display at
Iceland's National Museum in Reykjavik

Comparison between acute and chronic inflammation:

	Acute	Chronic
Causative agent	Pathogens, injured tissues	Persistent acute inflammation due to non-degradable pathogens, persistent foreign bodies, or autoimmune reactions
Major cells involved	Neutrophils, mononuclear cells (monocytes, macrophages)	Mononuclear cells (monocytes, macrophages, lymphocytes, plasma cells), fibroblasts
Primary mediators	Vasoactive amines, eicosanoids	IFN-γ and other cytokines, growth factors, reactive oxygen species, hydrolytic enzymes
Onset	Immediate	Delayed
Duration	Few days	Up to many months, or years
Outcomes	Resolution, abscess formation, chronic inflammation	Tissue destruction, fibrosis

wikipedia

Comparison between acute and chronic inflammation:

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wikipedia

Comparison between acute and chronic inflammation:

	Acute	Chronic
Primary mediators	Vasoactive amines, eicosanoids (In biochemistry, eicosanoids are signaling molecules made by oxygenation of twenty-carbon essential fatty acids, (EFAs).	IFN-γ and other cytokines, growth factors, reactive oxygen species, hydrolytic enzymes

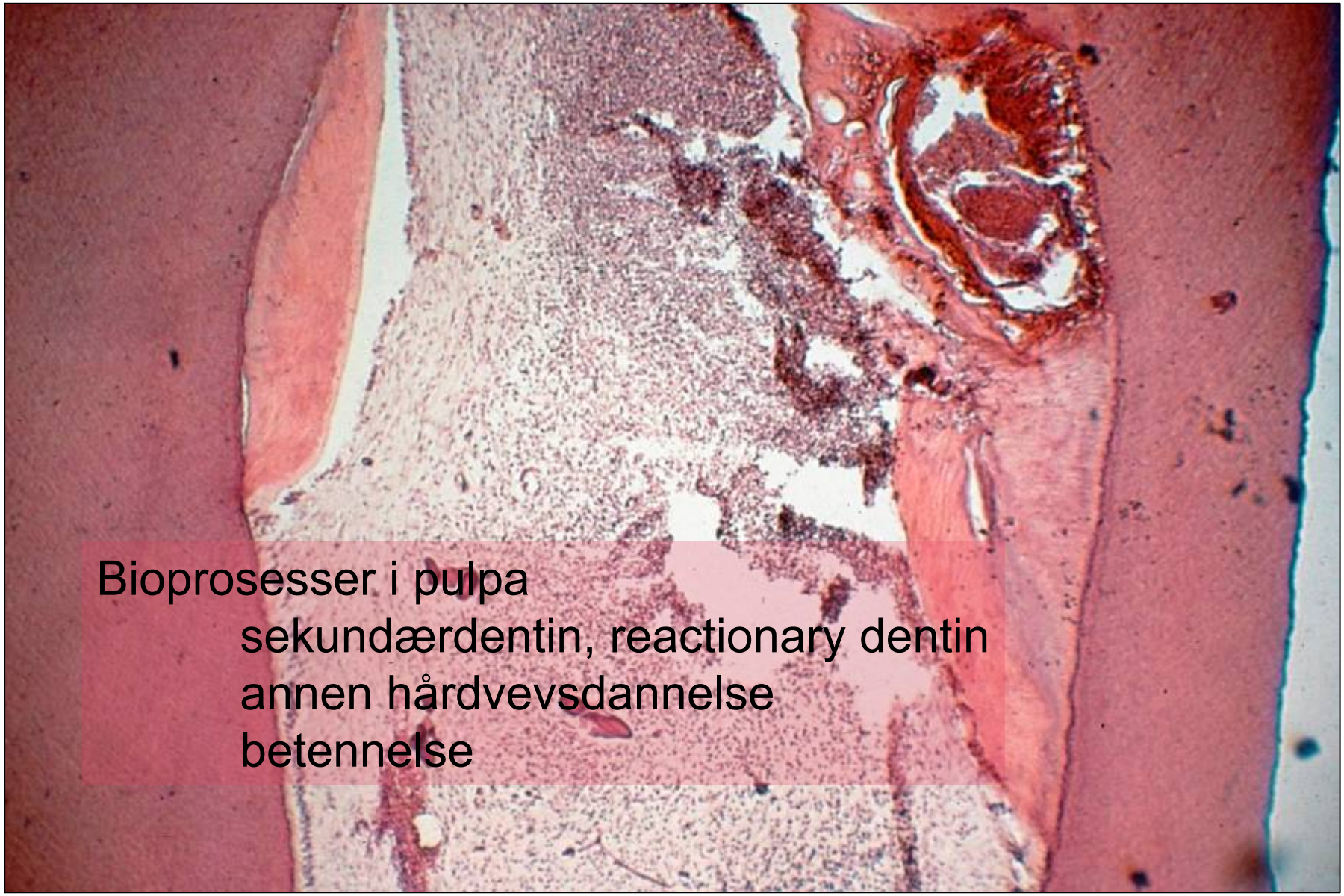
wikipedia

Name	Produced by	Description
Bradykinin	Kinin system	A vasoactive protein which is able to induce vasodilation, increase vascular permeability, cause smooth muscle contraction, and induce pain.
C3	Complement system	Cleaves to produce *C3a* and *C3b*. C3a stimulates histamine release by mast cells, thereby producing vasodilation. C3b is able to bind to bacterial cell walls and act as an opsonin, which marks the invader as a target for phagocytosis.
C5a	Complement system	Stimulates histamine release by mast cells, thereby producing vasodilation. It is also able to act as a chemoattractant to direct cells via chemotaxis to the site of inflammation.

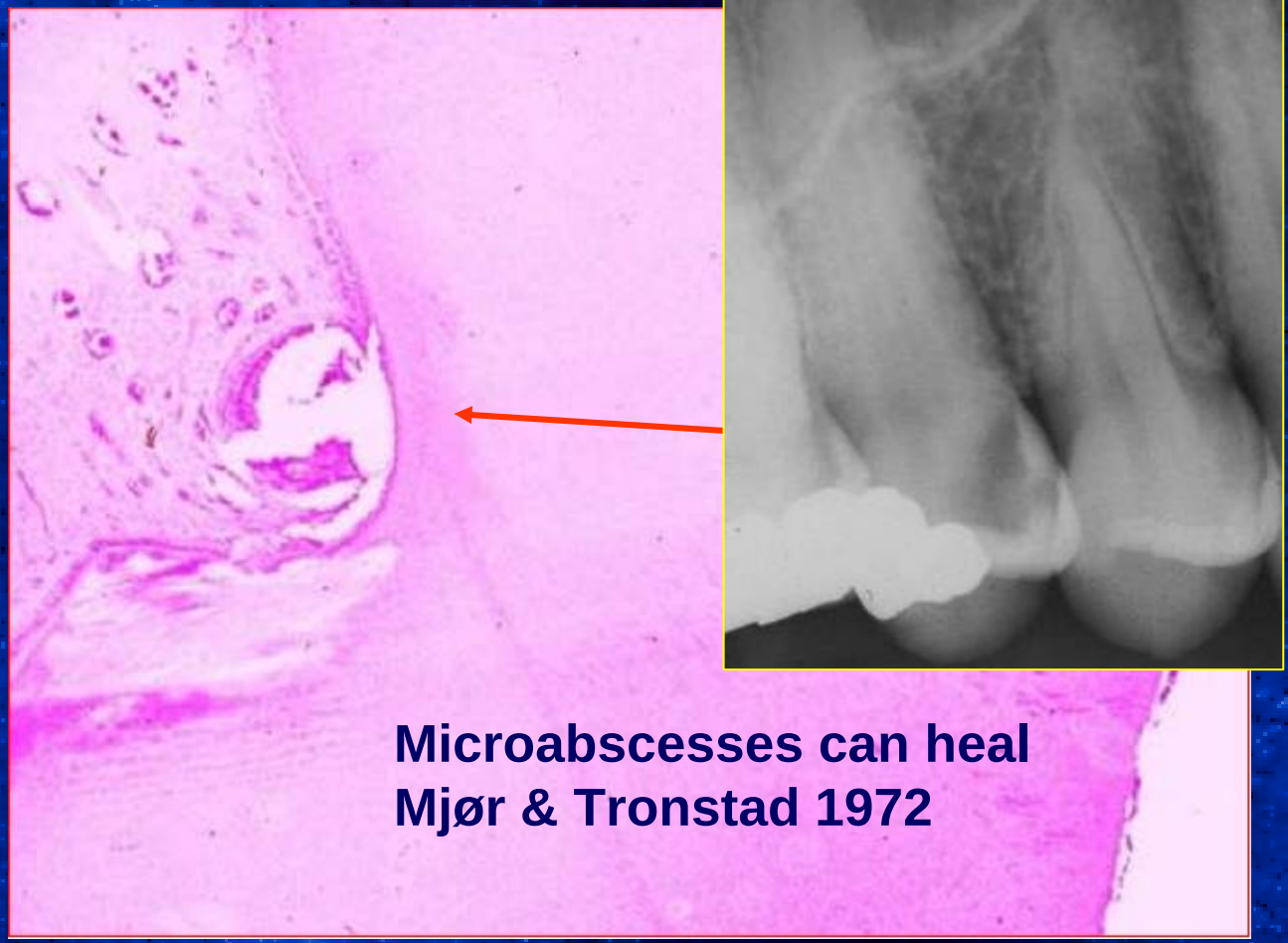
wikipedia

Name	Produced by	Description
Factor XII (Hageman Factor)	Liver	A protein which circulates inactively, until activated by collagen, platelets, or exposed basement membranes via conformational change. When activated, it in turn is able to activate three plasma systems involved in inflammation: the kinin system, fibrinolysis system, and coagulation system.
Membrane attack complex	Complement system	A complex of the complement proteins C5b, C6, C7, C8, and multiple units of C9. The combination and activation of this range of complement proteins forms the *membrane attack complex*, which is able to insert into bacterial cell walls and causes cell lysis with ensuing death.
Plasmin	Fibrinolysis system	Able to break down fibrin clots, cleave complement protein C3, and activate Factor XII.
Thrombin	Coagulation system	Cleaves the soluble plasma protein fibrinogen to produce insoluble fibrin, which aggregates to form a blood clot. Thrombin can also bind to cells via the PAR1 receptor to trigger several other inflammatory responses, such as production of chemokines and nitric oxide.

wikipedia


Bioprosesser i pulpa
sekundærdentin, reactionary dentin
annen hårdvevsdannelse
betennelse

Vital, inflamed: reversible-irreversible pulpitis



Microabscesses can heal
Mjør & Tronstad 1972

Haapasalo & Endal

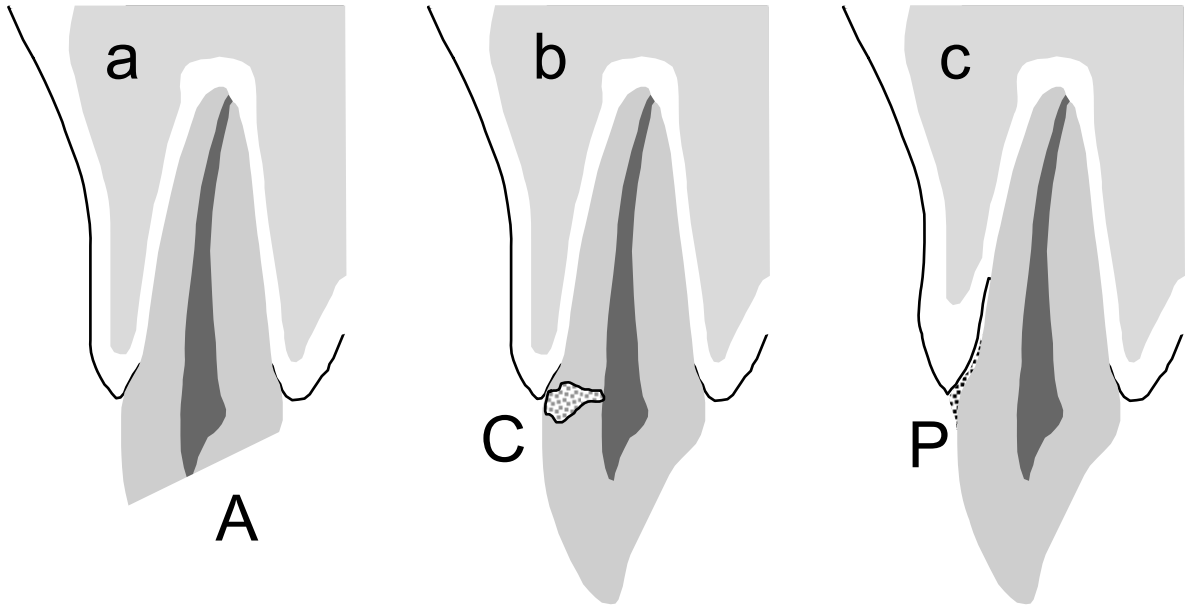
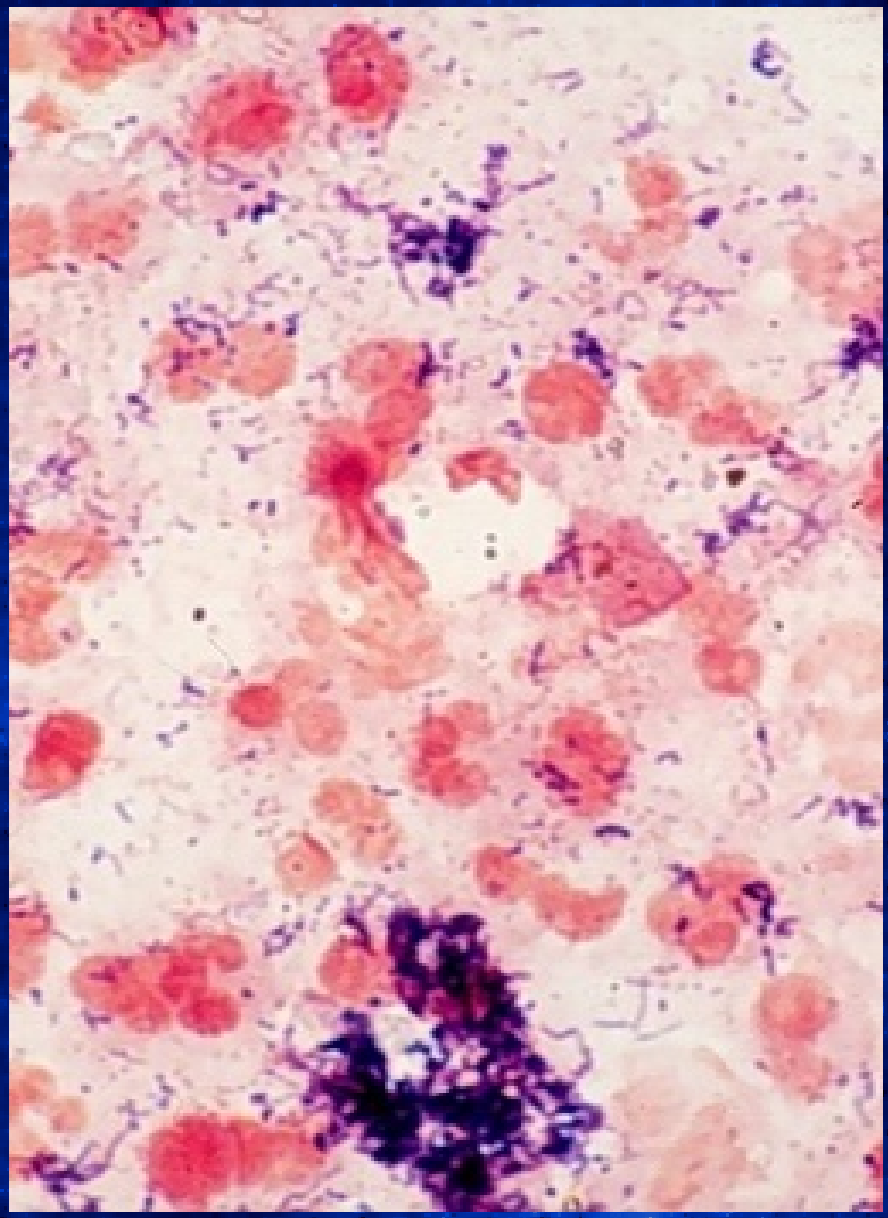
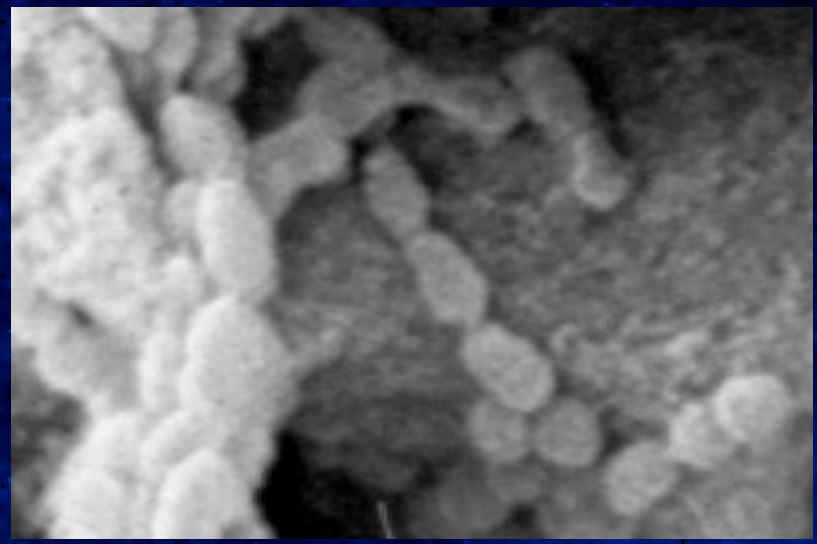


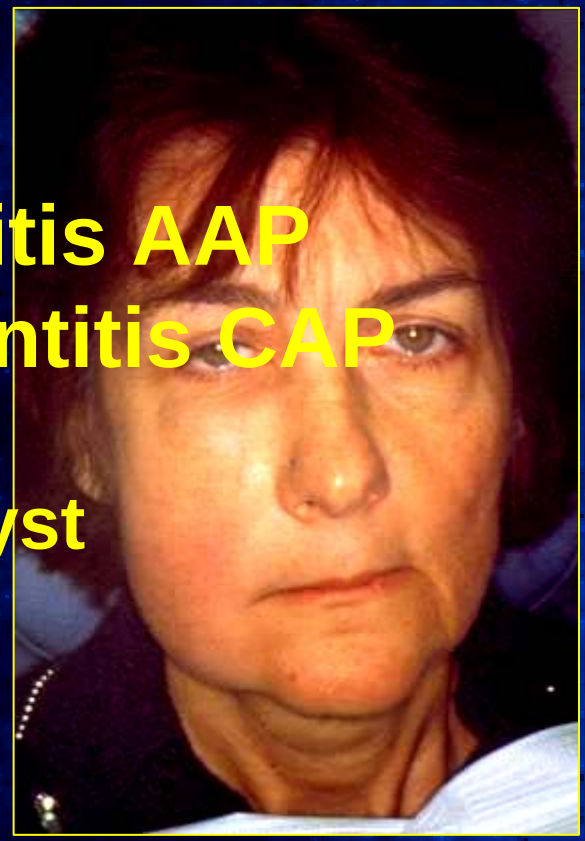
Fig. 1.3. Breaks in the muco-cutaneous barrier associated with teeth. (a) Attrition (A), abrasion or trauma exposes the pulp. (b) Dental caries (C) reaches the pulp with subsequent infection of the pulp and periapical tissues. (c) Dental plaque (P) penetrates the gingival cuff and bacteria invade the gingival and periodontal tissues.





Periapical diagnoses

- Transient AP
- Incipient AP
- Condensing AP
- Acute apical periodontitis AAP
- Chronic apical periodontitis CAP
  - Dental granuloma
  - Cyst: true cyst or bay cyst
- Exacerbating CAP



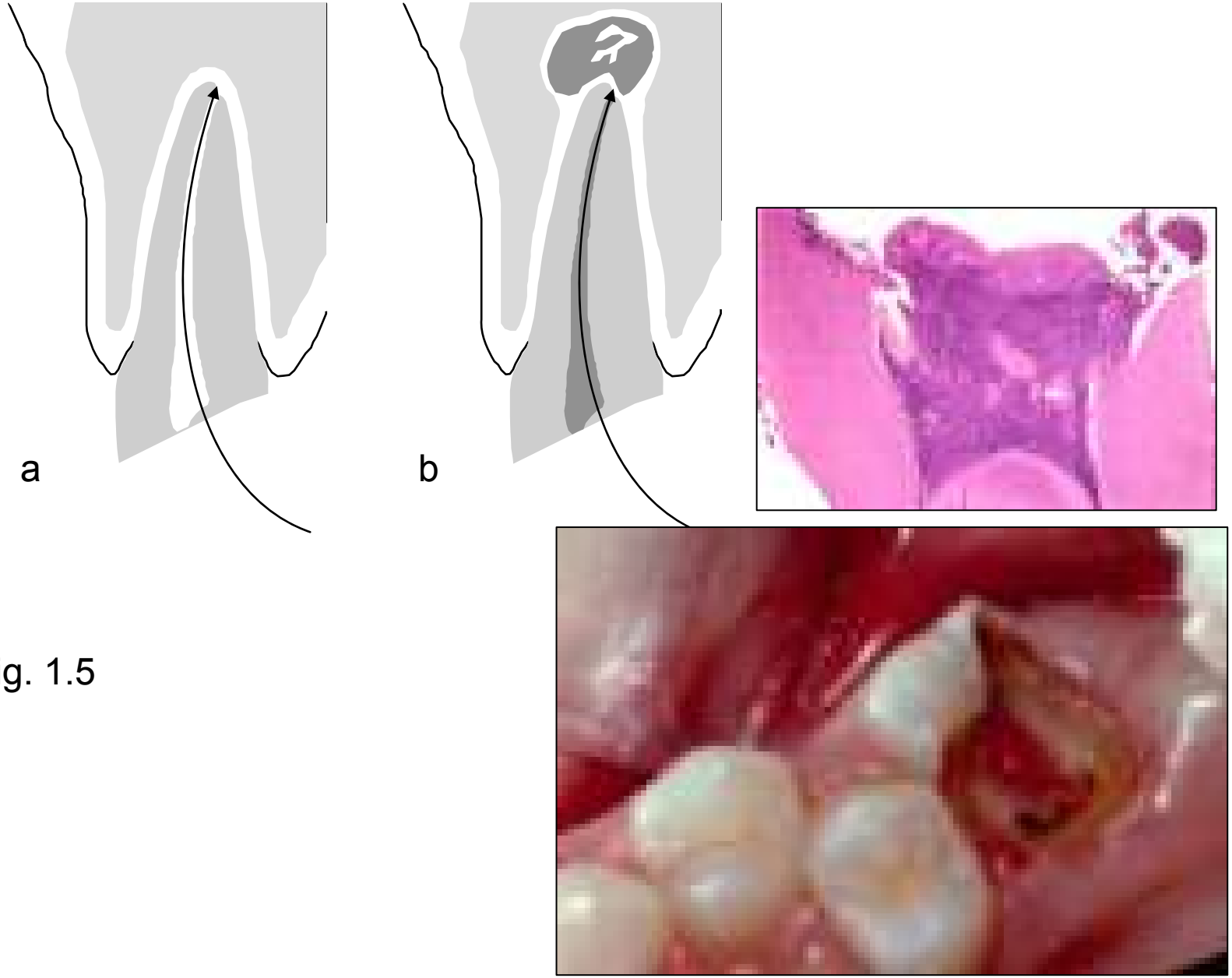
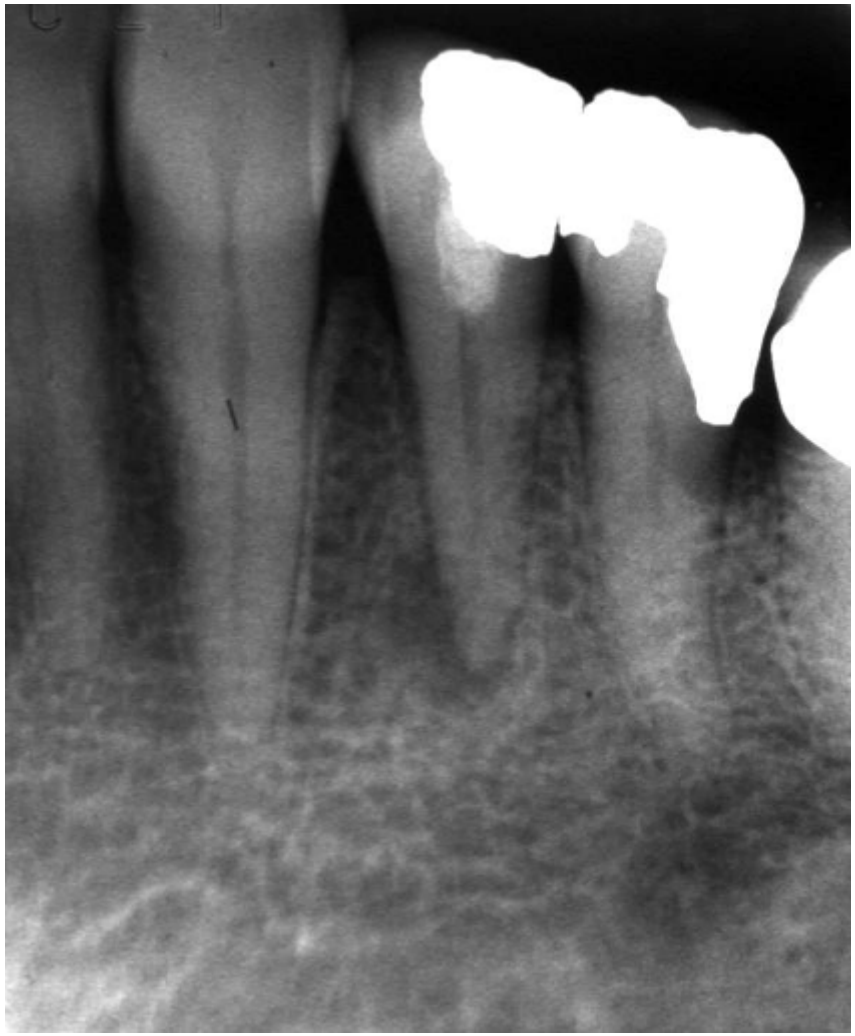
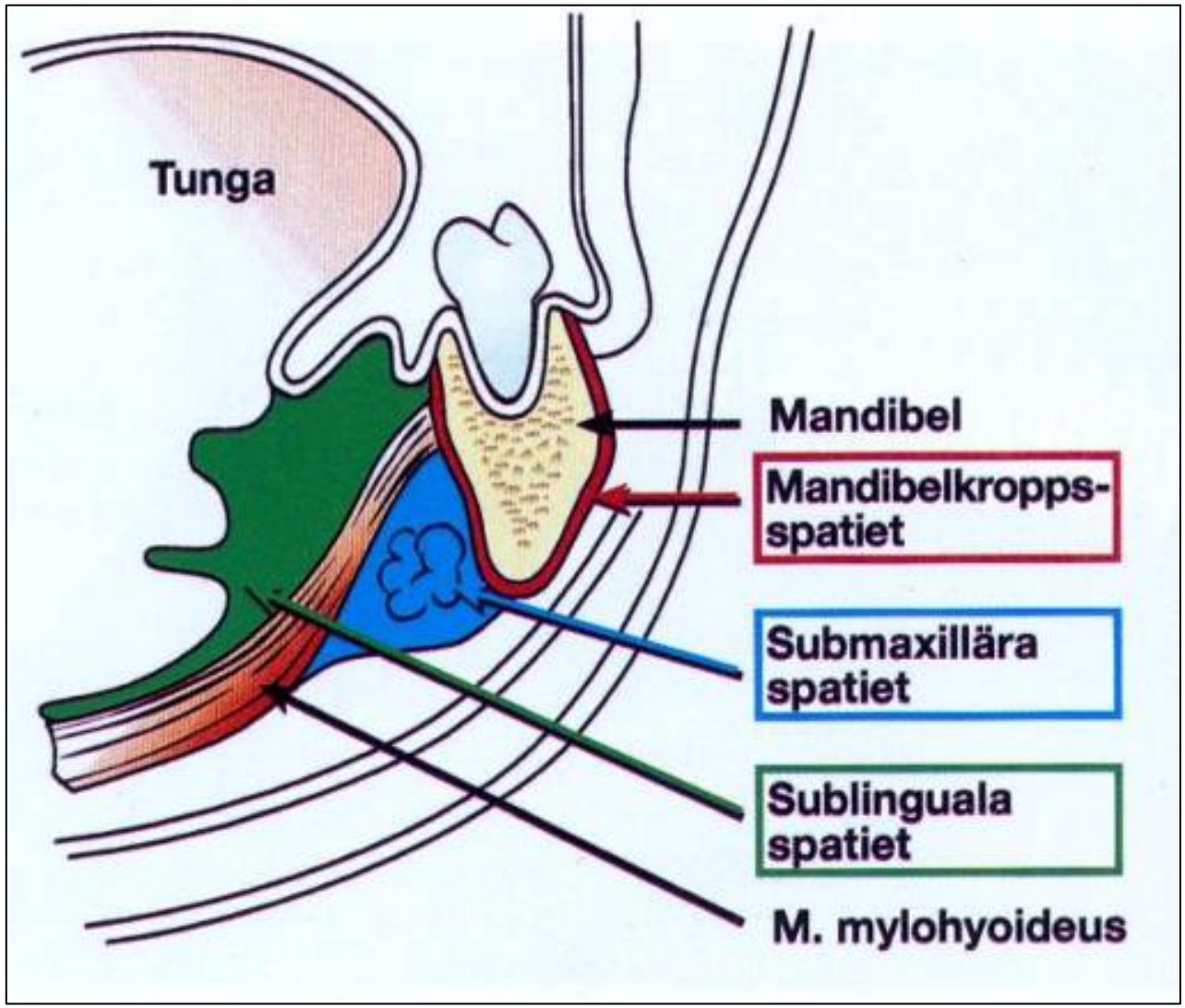
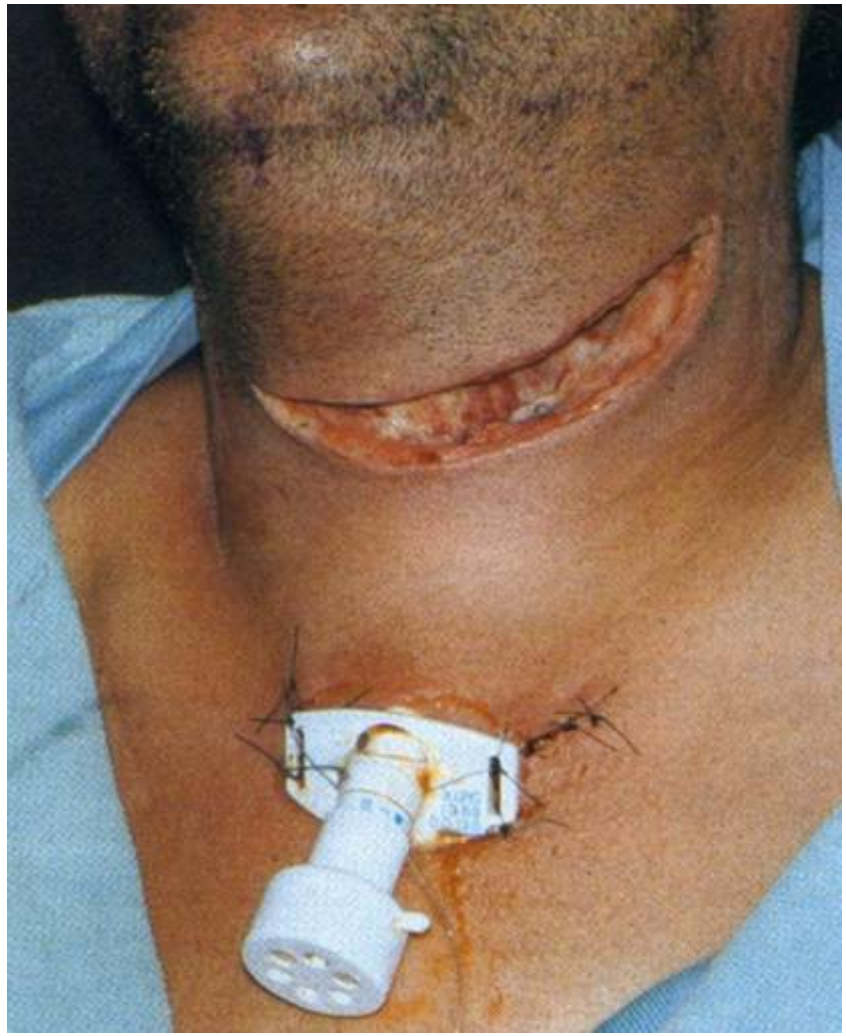


Fig. 1.5

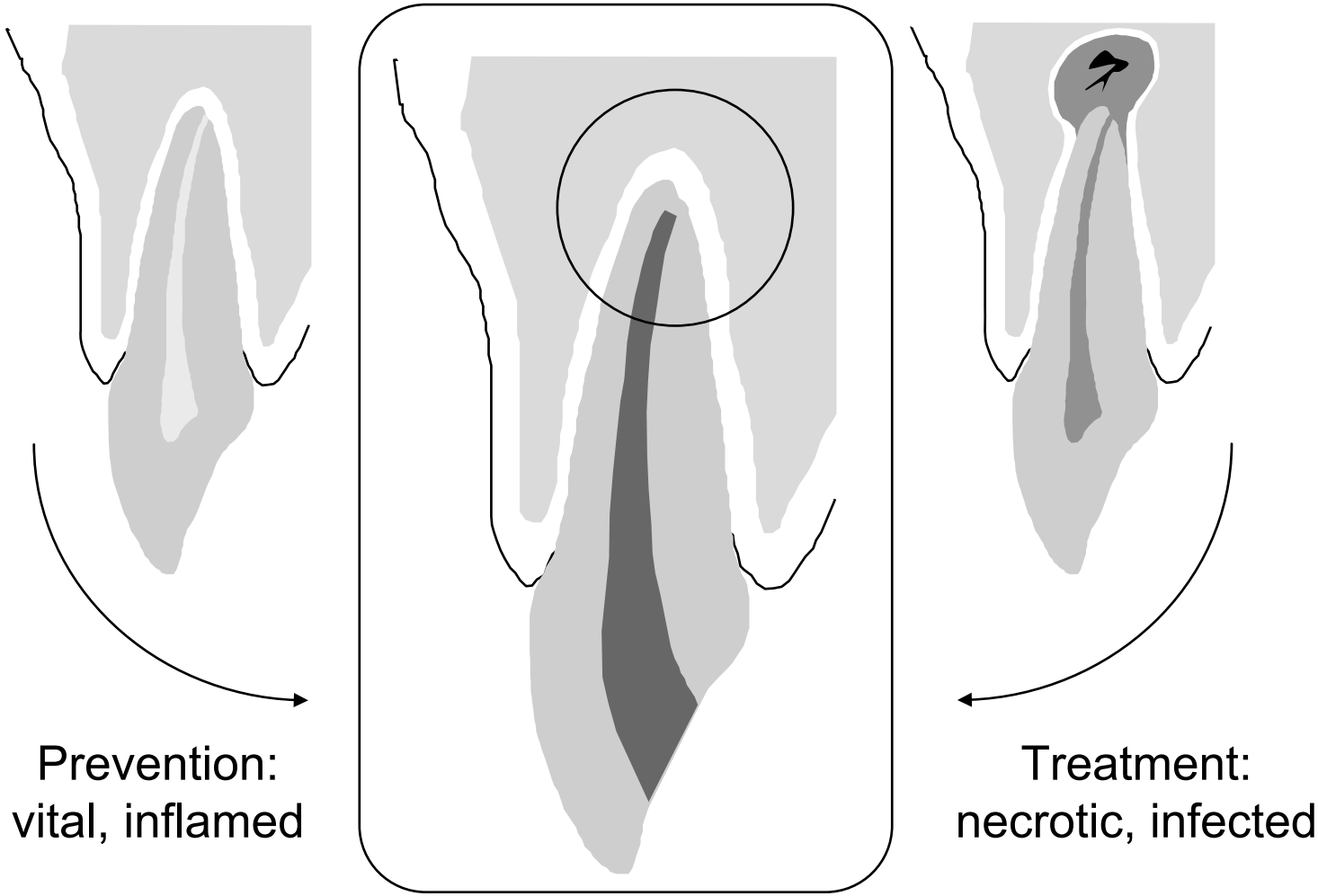




Adielsson et al 2003



Adielsson et al 2003

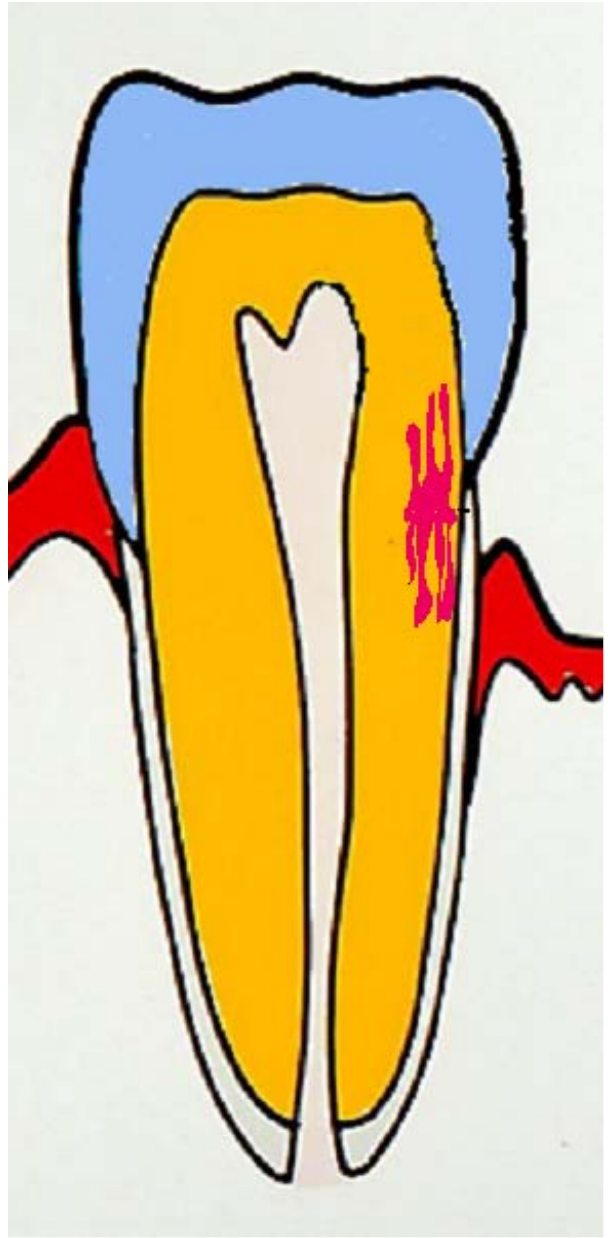


Success
= absence of apical periodontitis:
clinically, radiographically, histologically

Fig. 1.1

Tooth characteristics in relation to endodontic diagnosis

- ✓ Caries
- ✓ Erosion/abrasion/ attrition
- ✓ Defective fillings/margins
- ✓ Tooth fractures



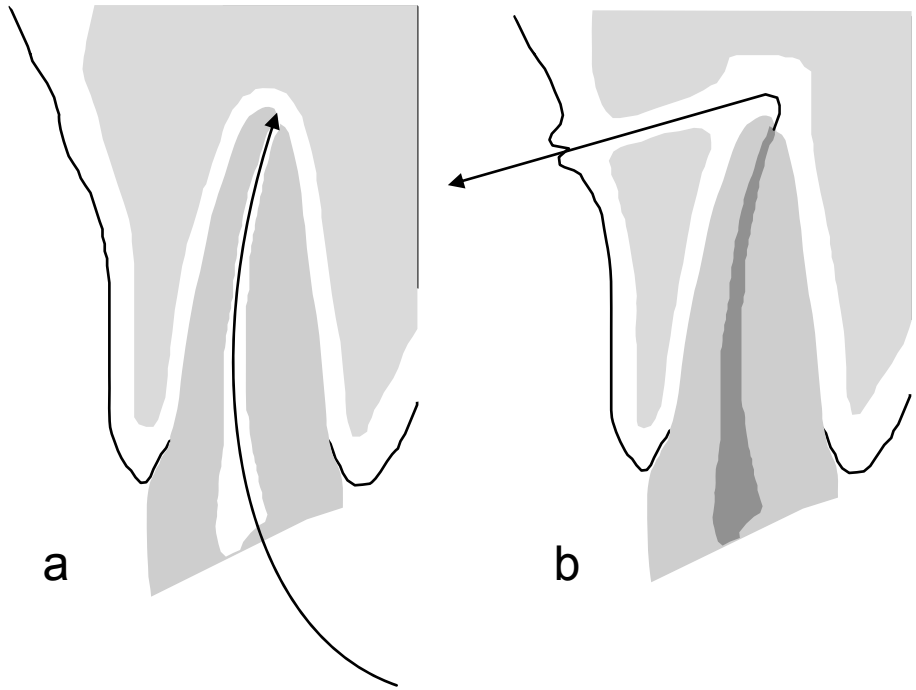


Fig. 1.6

Fig. 3.1. Pathways of periapical inflammation and bone destruction.

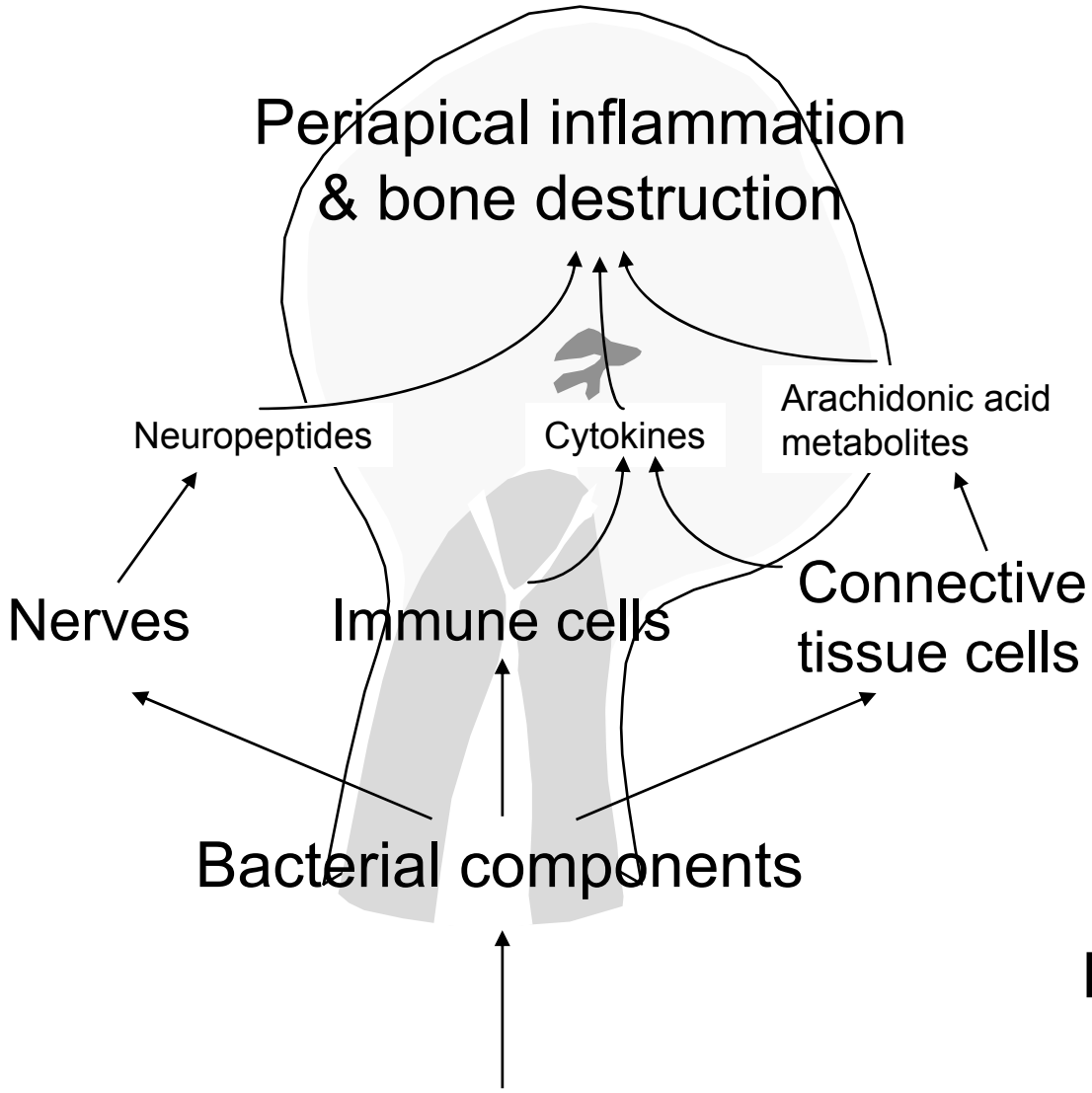
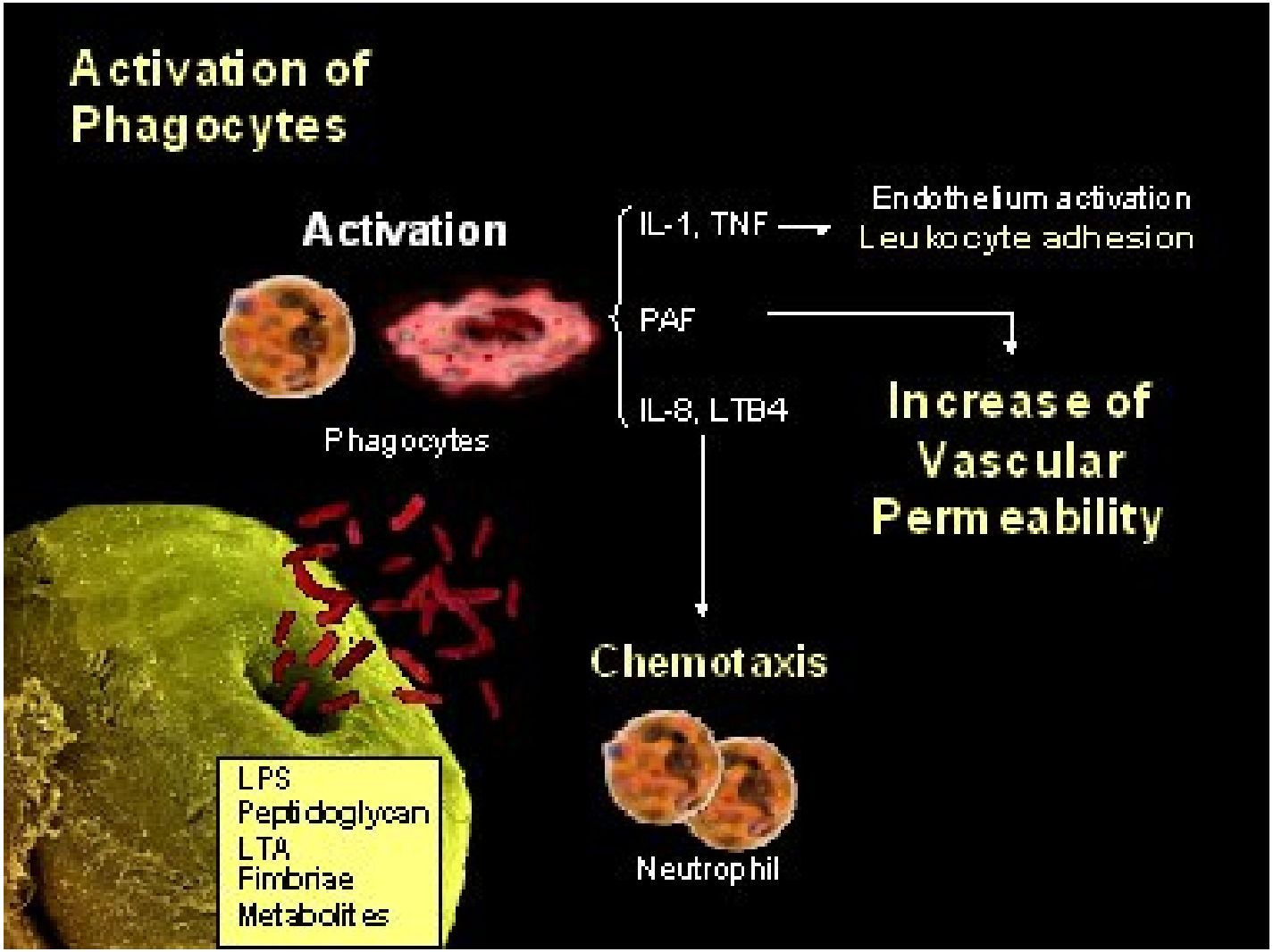
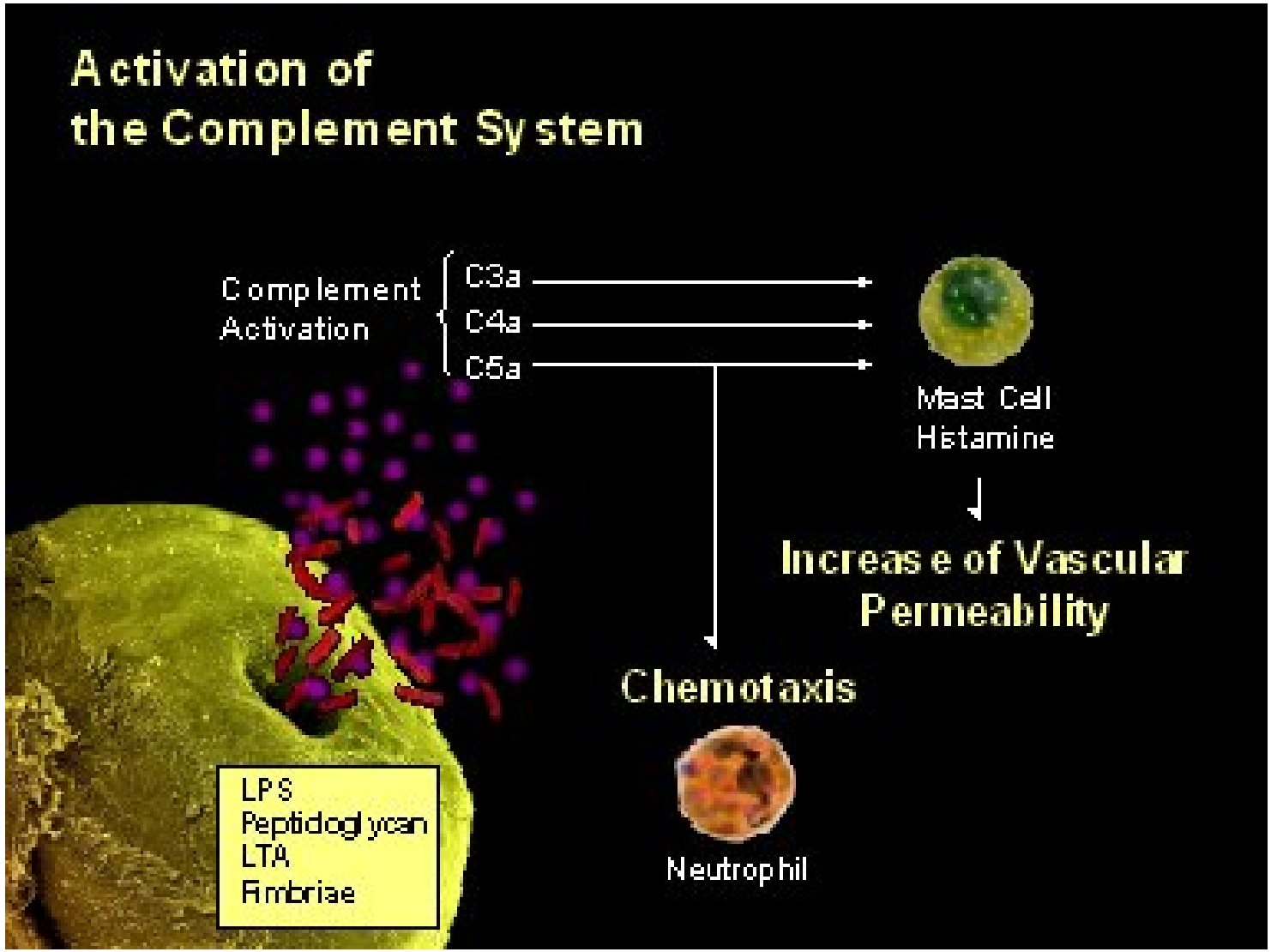


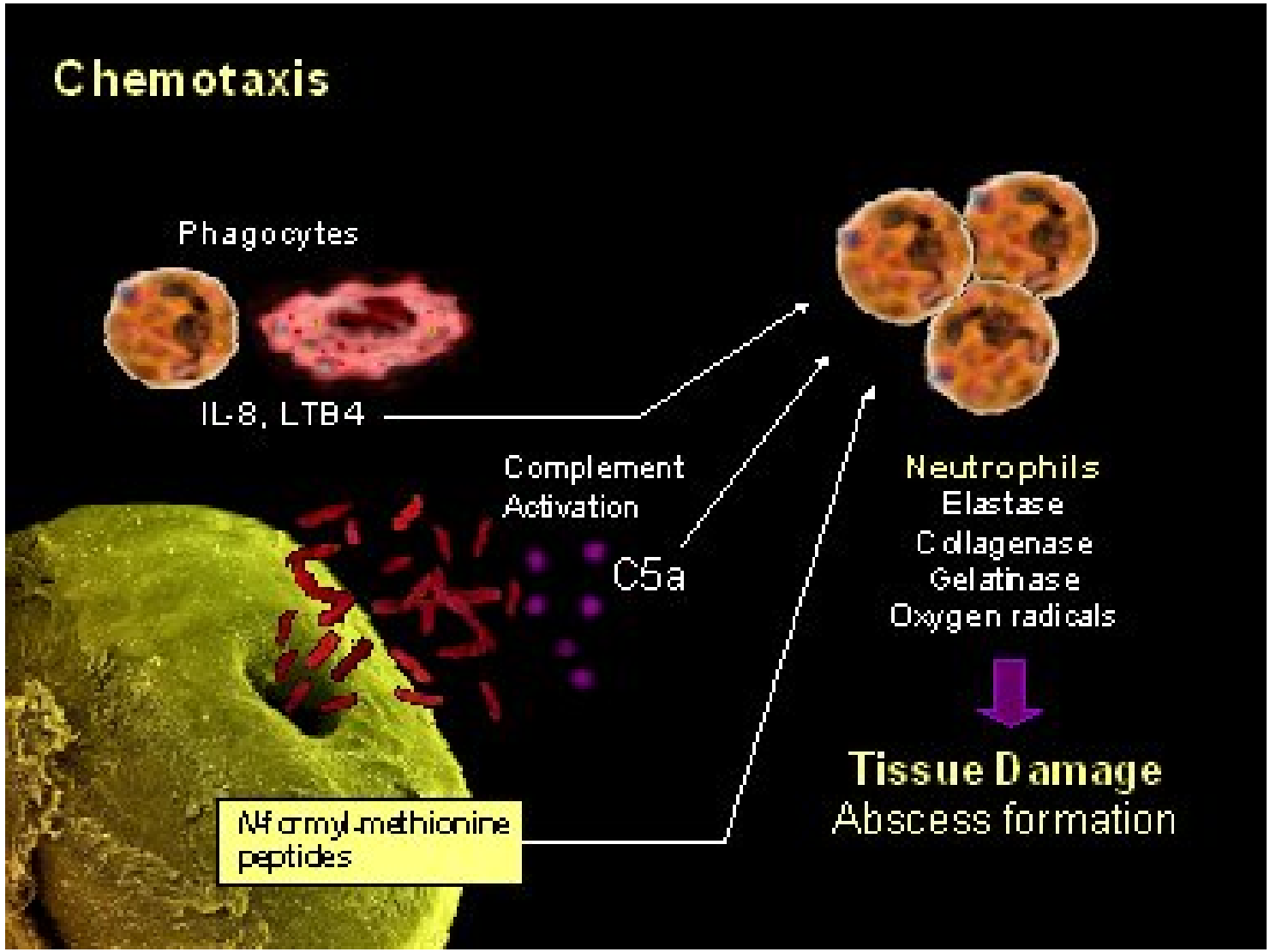
Fig. 3.1



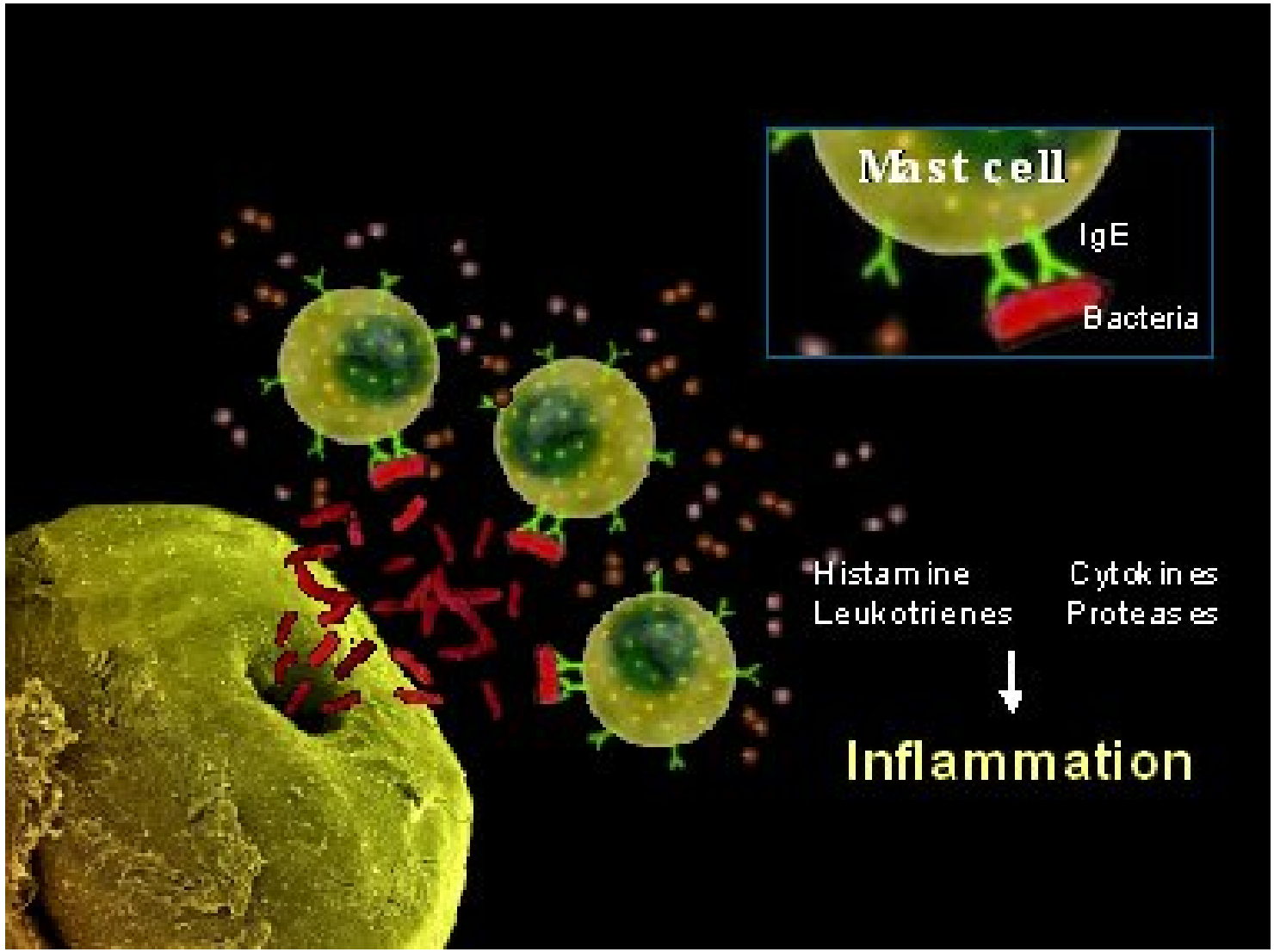
Acute aspects; Siqueira & Barnett 2004



Acute aspects; Siqueira & Barnett 2004



Acute aspects; Siqueira & Barnett 2004



Acute aspects; Siqueira & Barnett 2004

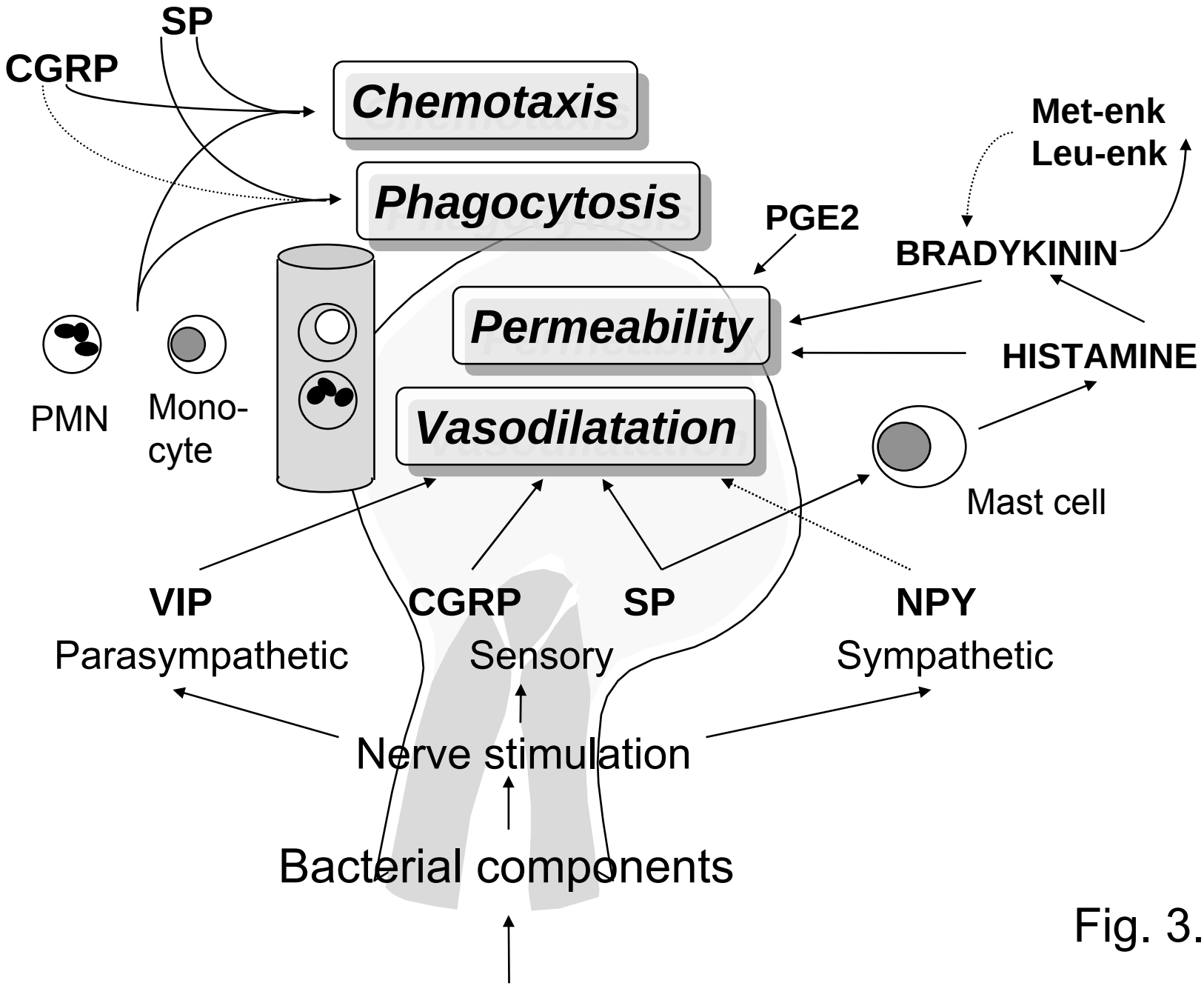
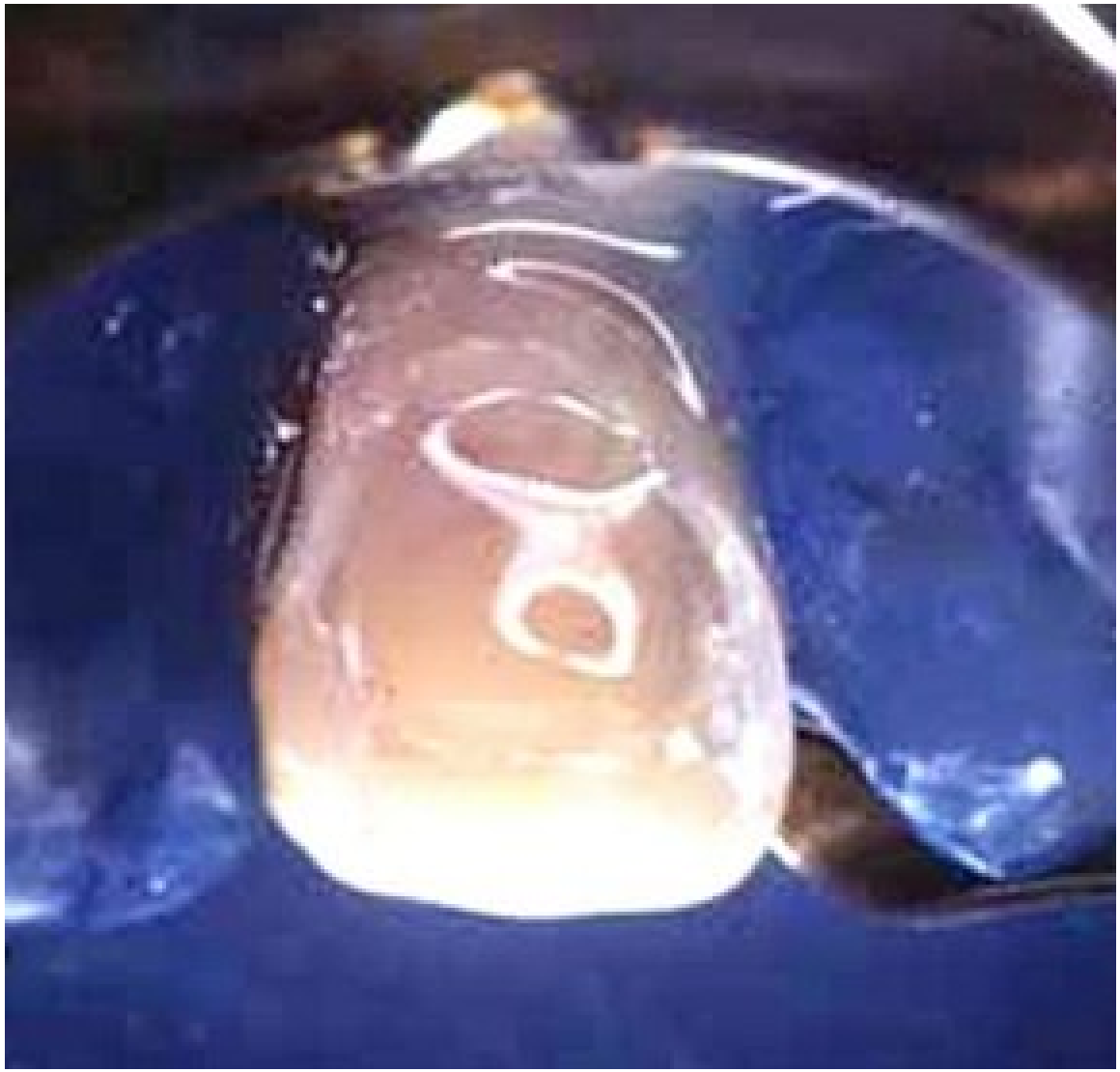
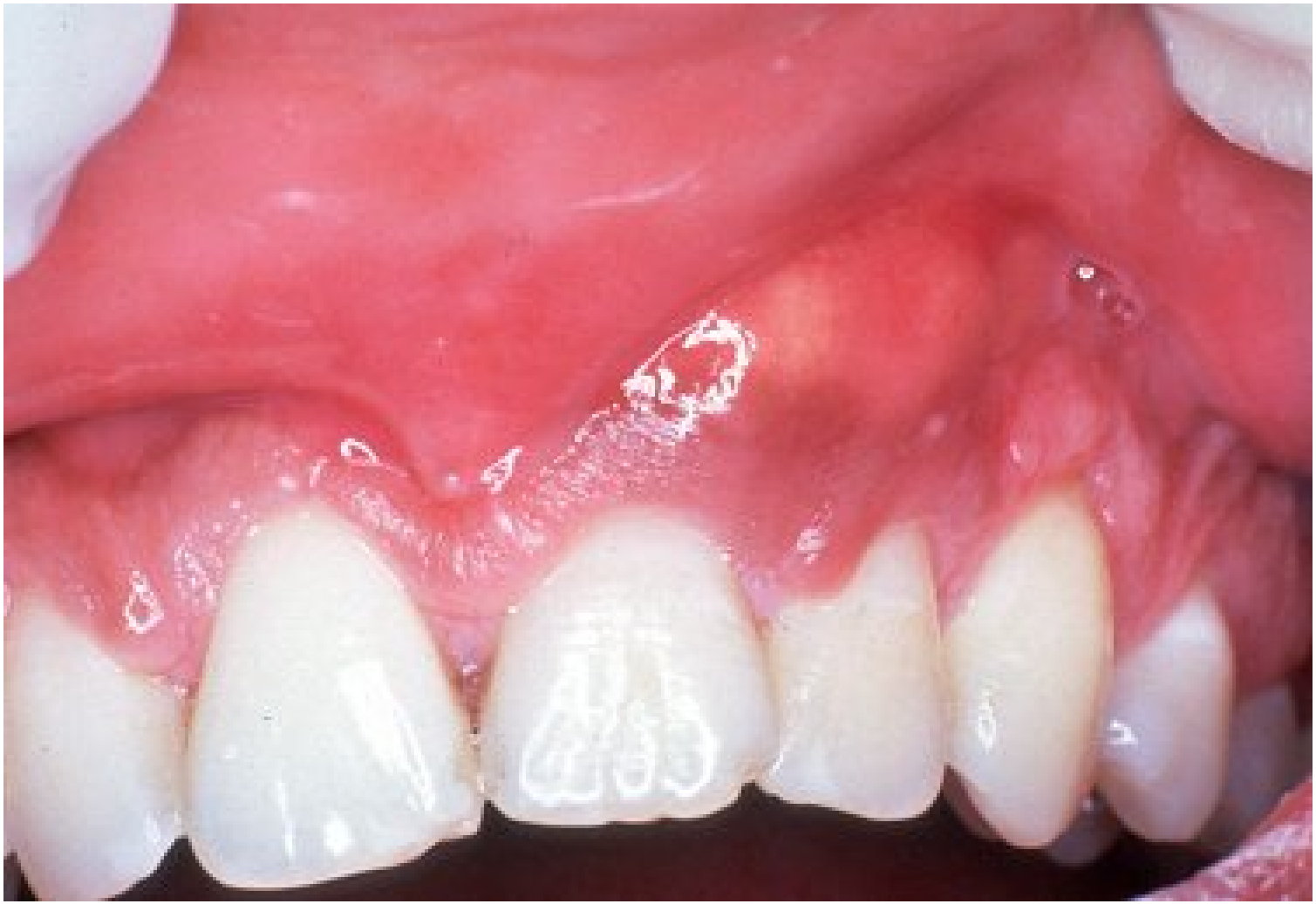

SP
CGRP
Chemotaxis
Phagocytosis
Met-enk
Leu-enk
PGE2
BRADYKININ
Permeability
HISTAMINE
PMN
Mono-
cyte
Vasodilatation
Mast cell
VIP
Parasympathetic
CGRP
Sensory
SP
NPY
Sympathetic
Nerve stimulation
Bacterial components

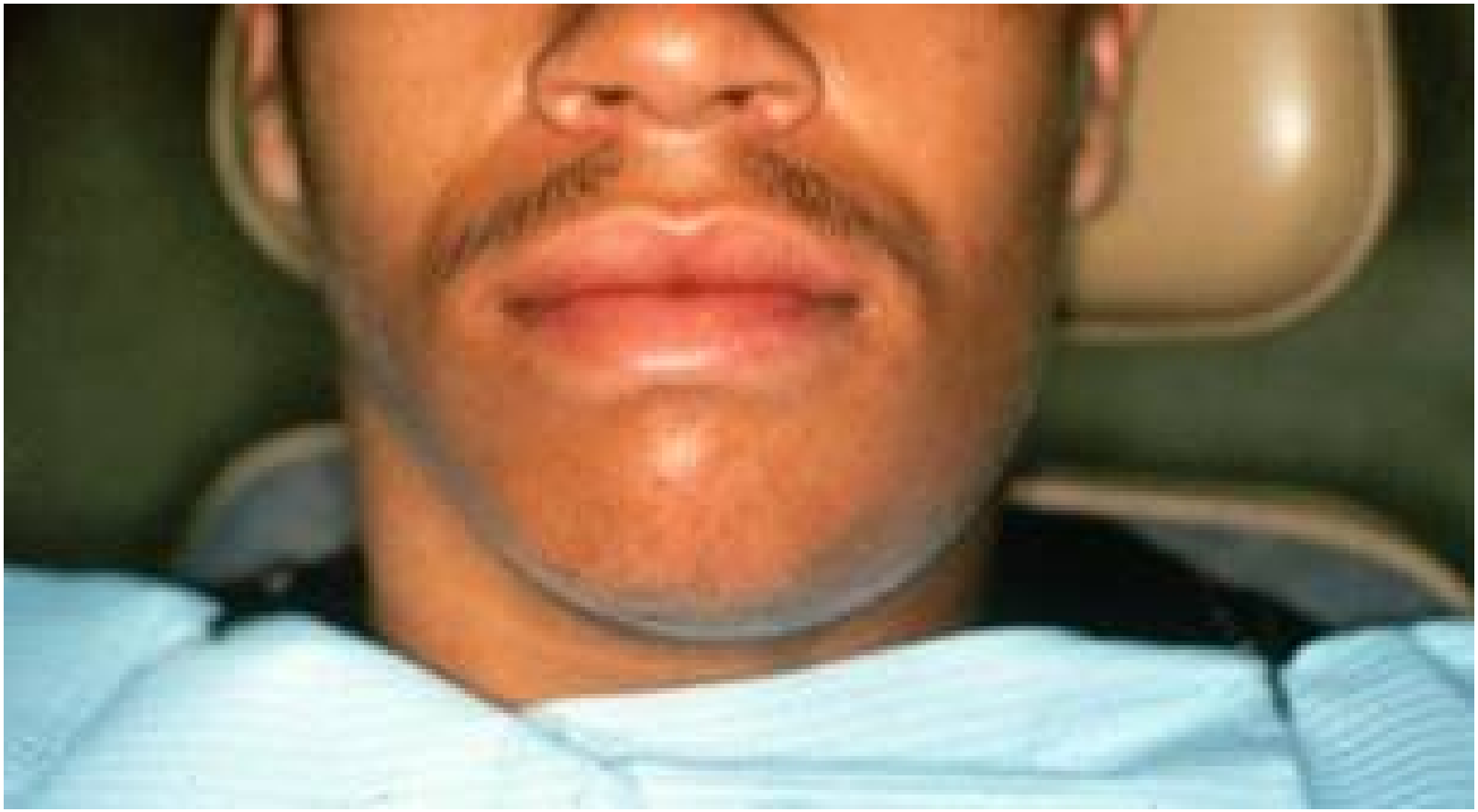
Fig. 3.6



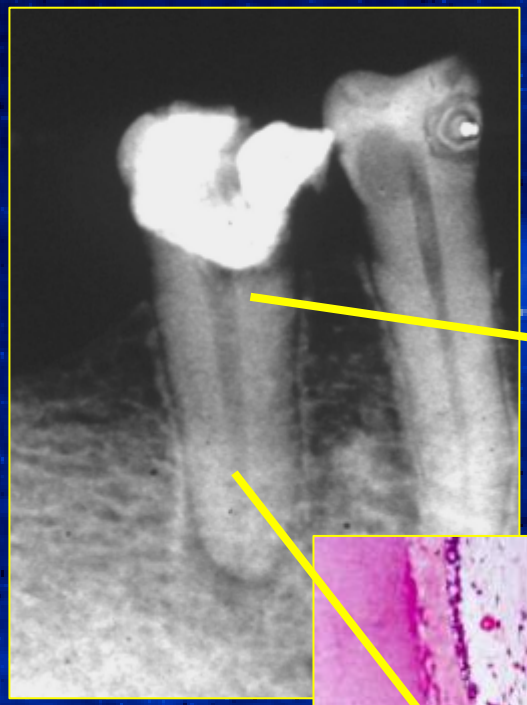
Siqueira & Barnett 2004

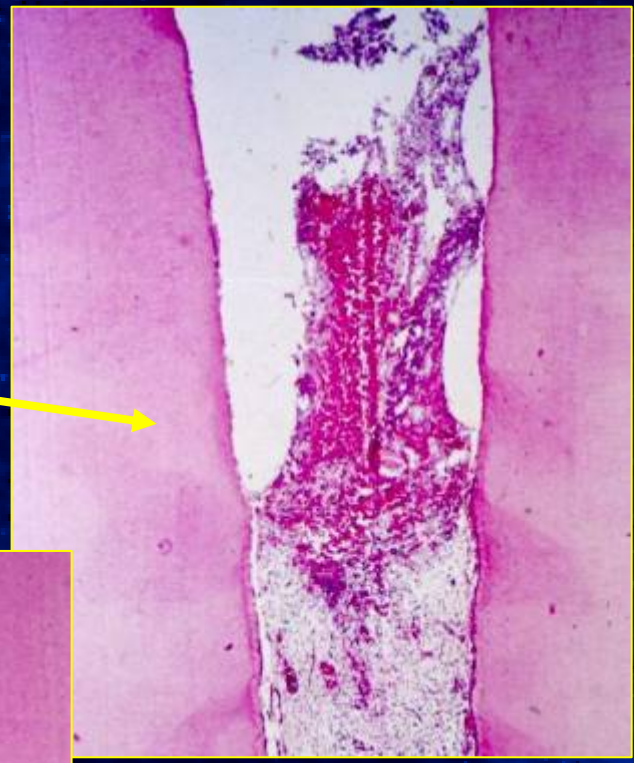


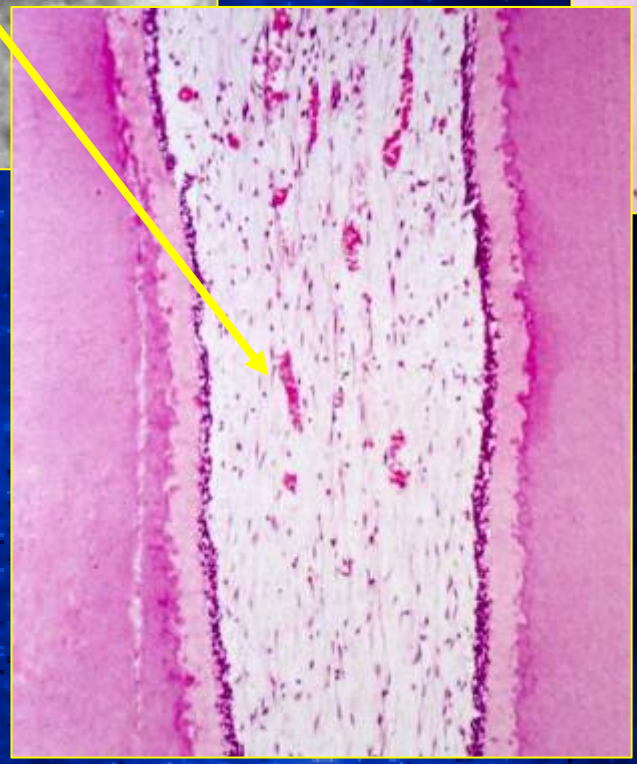
Siqueira & Barnett 2004



Siqueira & Barnett 2004







Infection, not necrosis, is essential for development of AP

Ørstavik, Essential Endodontology 1998; courtesy of Lambjerg Hansen

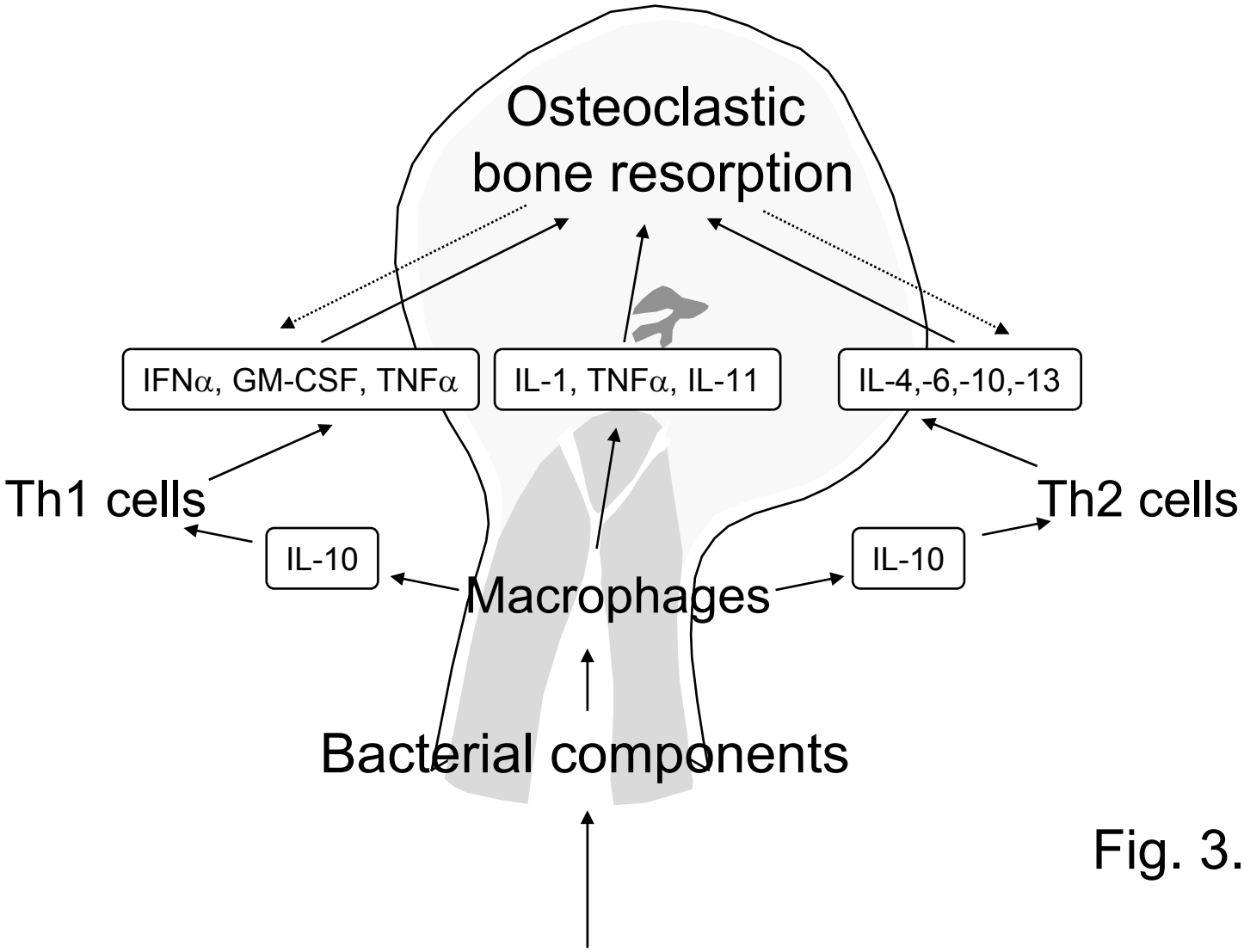
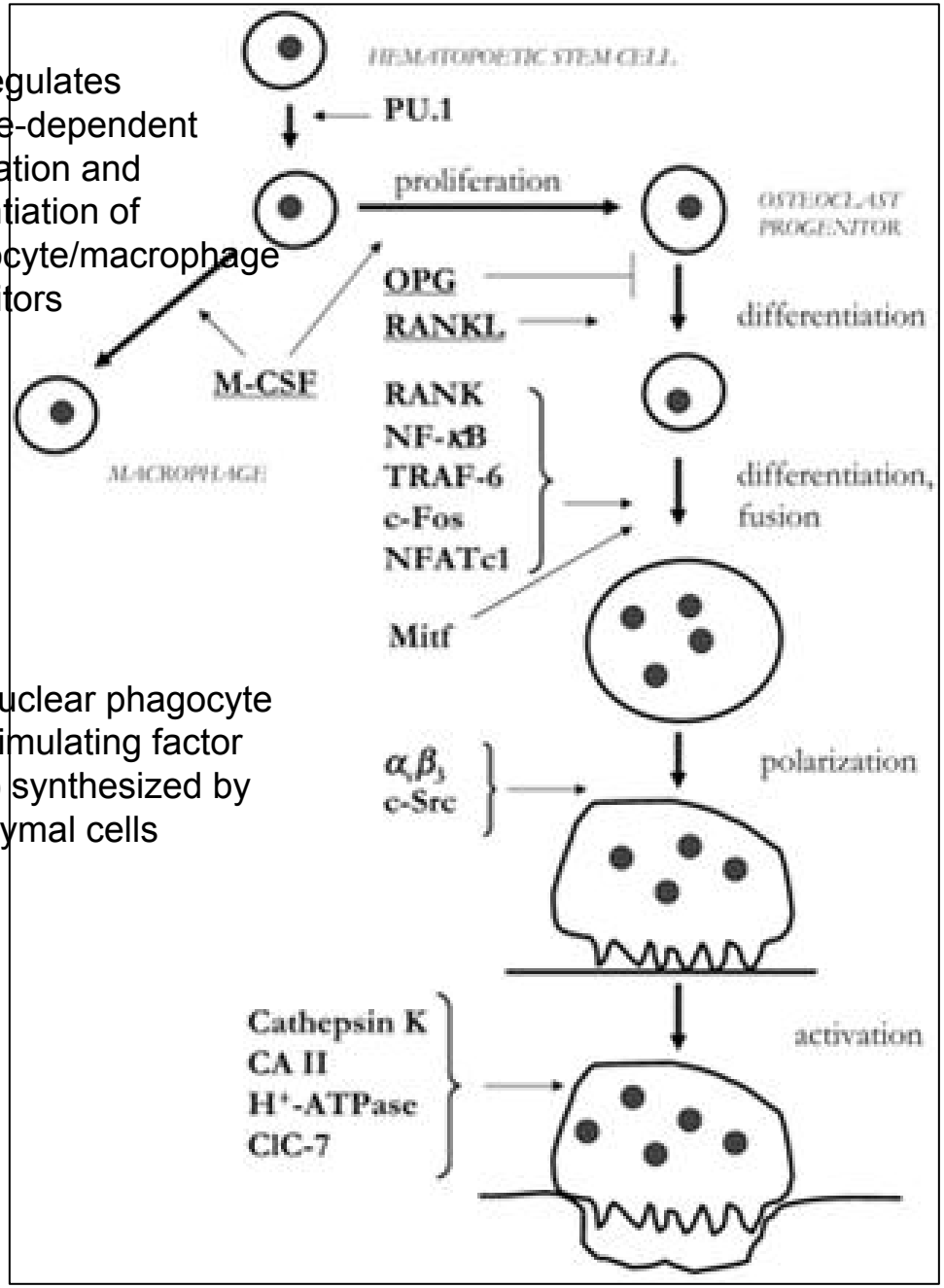

Osteoclastic
bone resorption
IFNα, GM-CSF, TNFα
IL-1, TNFα, IL-11
IL-4,-6,-10,-13
Th1 cells
Th2 cells
IL-10
IL-10
Macrophages
Bacterial components

Fig. 3.7

The resorptive process

- Denudation:
  - Cementum
  - Predentin
- Remodelling:
  - Deposition
  - resorption
- Infectious/pathological
  - Internal inflammatory
  - External inflammatory
- Physiological/protective
  - Pressure induced
  - Surface repair
  - Replacement/ankylosis

PU.1 regulates cytokine-dependent proliferation and differentiation of granulocyte/macrophage progenitors



A mononuclear phagocyte colony-stimulating factor (M-CSF) synthesized by mesenchymal cells

Receptor activator of nuclear factor- B ligand (RANKL) is a critical cytokine for osteoclast differentiation and activation and an essential regulator of osteoblast-osteoclast cross-talks (4). RANKL activates its receptor RANK, which is located on osteoclastic lineage cells, and this interaction is prevented by osteoprotegerin (OPG), which acts as an endogenous receptor antagonist and blocks the effects of RANKL (4). While RANKL enhances bone resorption and bone loss and promotes osteoporosis, OPG has opposite effects (5).

Crit Rev Oral Biol Med. 2004;15(2):64-81. NEW MOLECULES IN THE TUMOR NECROSIS FACTOR LIGAND AND RECEPTOR SUPERFAMILIES WITH IMPORTANCE FOR PHYSIOLOGICAL AND PATHOLOGICAL BONE RESORPTION. Lerner UH.

Hvordan oppstår odonto/osteo-klaster?

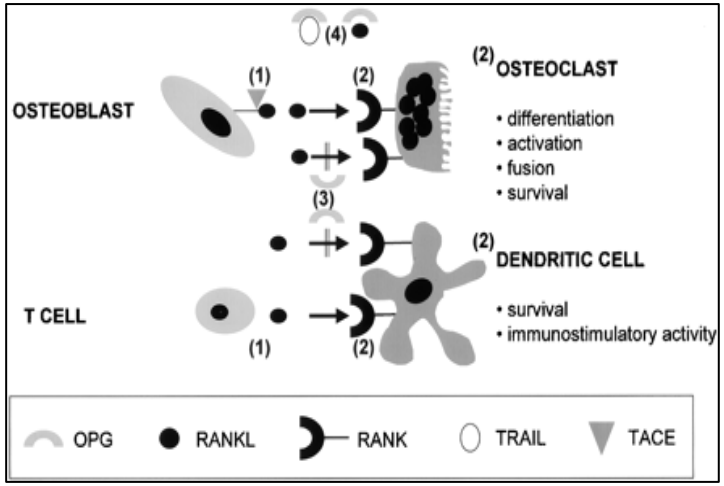
- Osteoclasts formation requires the presence of RANK ligand (receptor activator of nuclear factor κβ) and M-CSF (Macrophage colony-stimulating factor). These membrane bound proteins are produced by neighbouring stromal cells and osteoblasts; thus requiring direct contact between these cells and osteoclast precursors.
- M-CSF acts through its receptor on the osteoclast [precursor], c-fms (colony stimulating factor 1 receptor), a transmembrane tyrosine kinase-receptor, leading to secondary messenger activation of tyrosine kinase Src. Both of these molecules are necessary for osteoclastogenesis and are widely involved in the differentiation of monocyte/macrophage derived cells.

http://en.wikipedia.org/wiki/Osteoclast; 2007-06-21

Hvordan oppstår odonto/osteo-klaster?

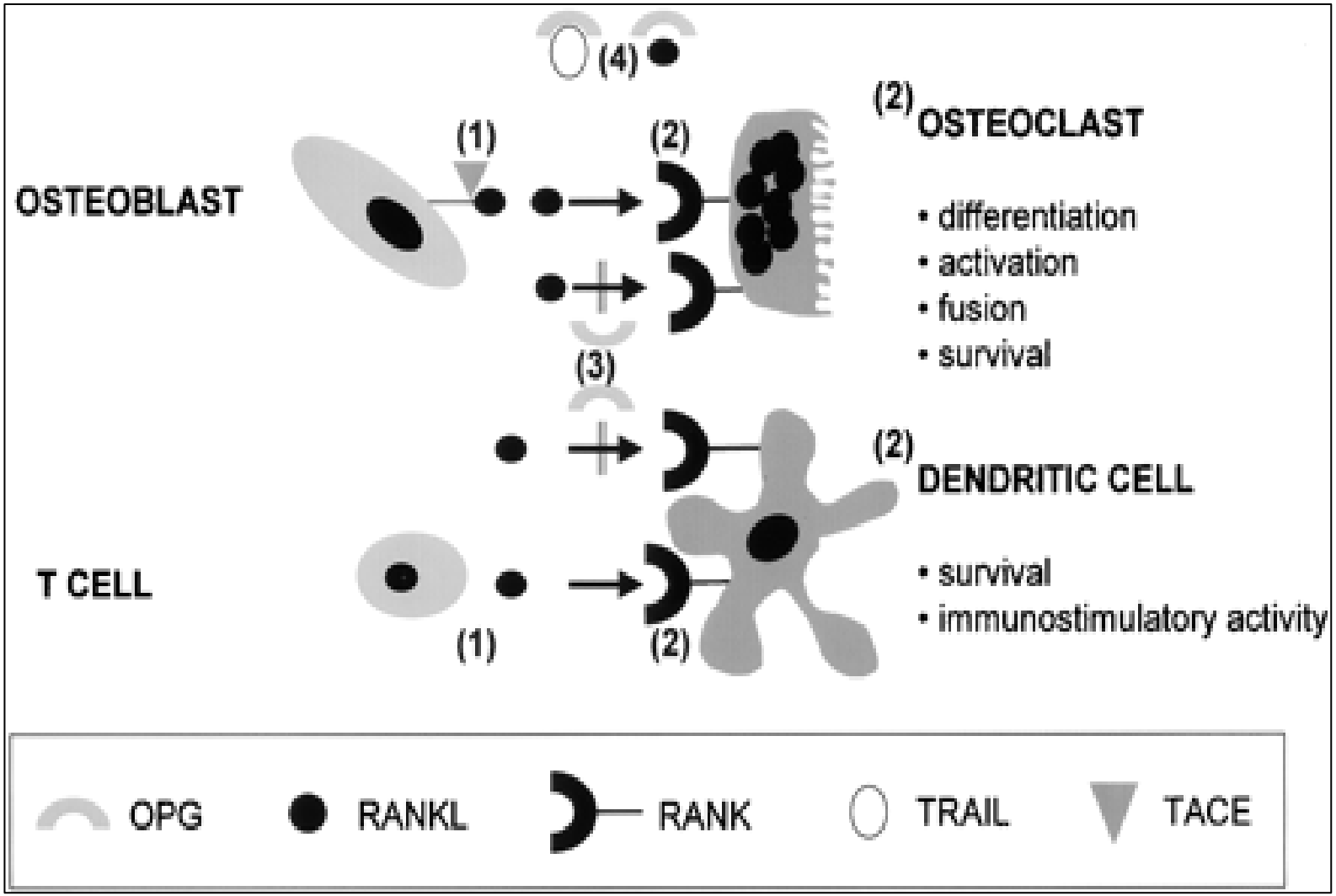
- RANKL is a member of the tumour necrosis family (TNF), and is essential in osteoclastogenesis. RANKL knockout mice exhibit a phenotype of osteopetrosis and defects of tooth eruption, along with an absence or deficiency of osteoclasts. RANKL activates NF-κβ (nuclear factor-κβ) and NFATc1 (nuclear factor of activated t cells, cytoplasmic, calcineurin-dependent 1) through RANK. NF-κβ activation is stimulated almost immediately after RANKL-RANK interaction occurs, and is not upregulated. NFATc1 stimulation, however, begins ~24-48 hours after binding occurs and its expression has been shown to be RANKL dependent.
- Osteoclast differentiation is inhibited[/regulated] by osteoprotegerin (OPG), which binds to RANKL thereby preventing interaction with RANK.

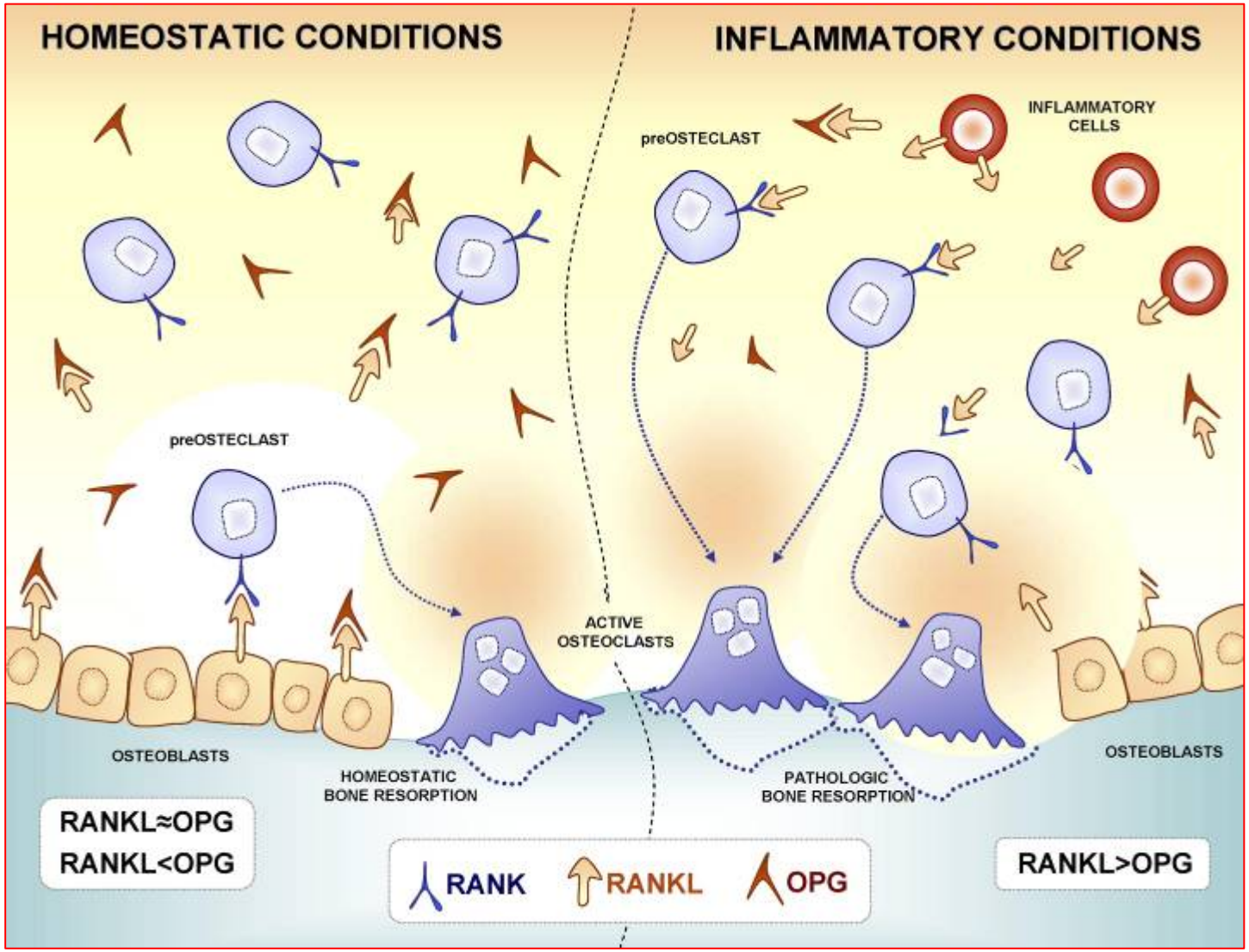
http://en.wikipedia.org/wiki/Osteoclast; 2007-06-21



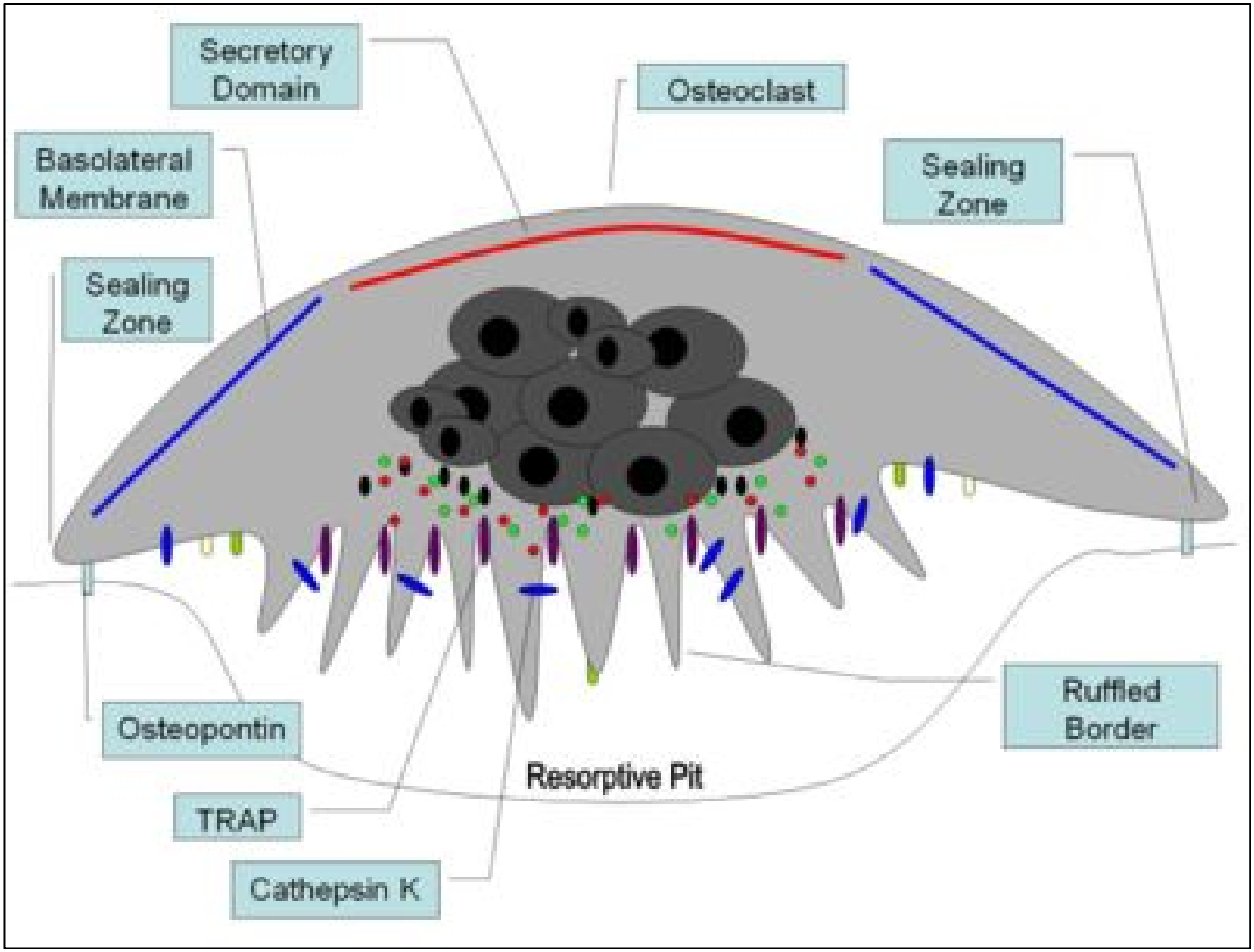
Schoppet M, Preissner KT, Hofbauer LC. RANK ligand and osteoprotegerin: paracrine regulators of bone metabolism and vascular function. Arterioscler Thromb Vasc Biol. 2002 Apr 1;22(4):549-53. Review.

Figure 2. Mode of action and biological effects of RANKL, RANK, and OPG on bone metabolism and the immune system. (1) RANKL is expressed by osteoblastic lineage cells (cell-bound RANKL) and activated T lymphocytes (soluble RANKL). A truncated ectodomain form of RANKL is derived from the cell-bound form after cleavage by the enzyme TACE. (2) All three RANKL variants stimulate their specific receptor, RANK, which is located on osteoclastic and dendritic cells and thus modulate various biological functions. (3) OPG is secreted by osteoblastic lineage and other cells and acts as a soluble receptor antagonist which neutralizes RANKL (black), and thus, prevents RANKL-RANK interaction.(4) OPG also blocks the pro-apoptotic cytokine TRAIL (white).

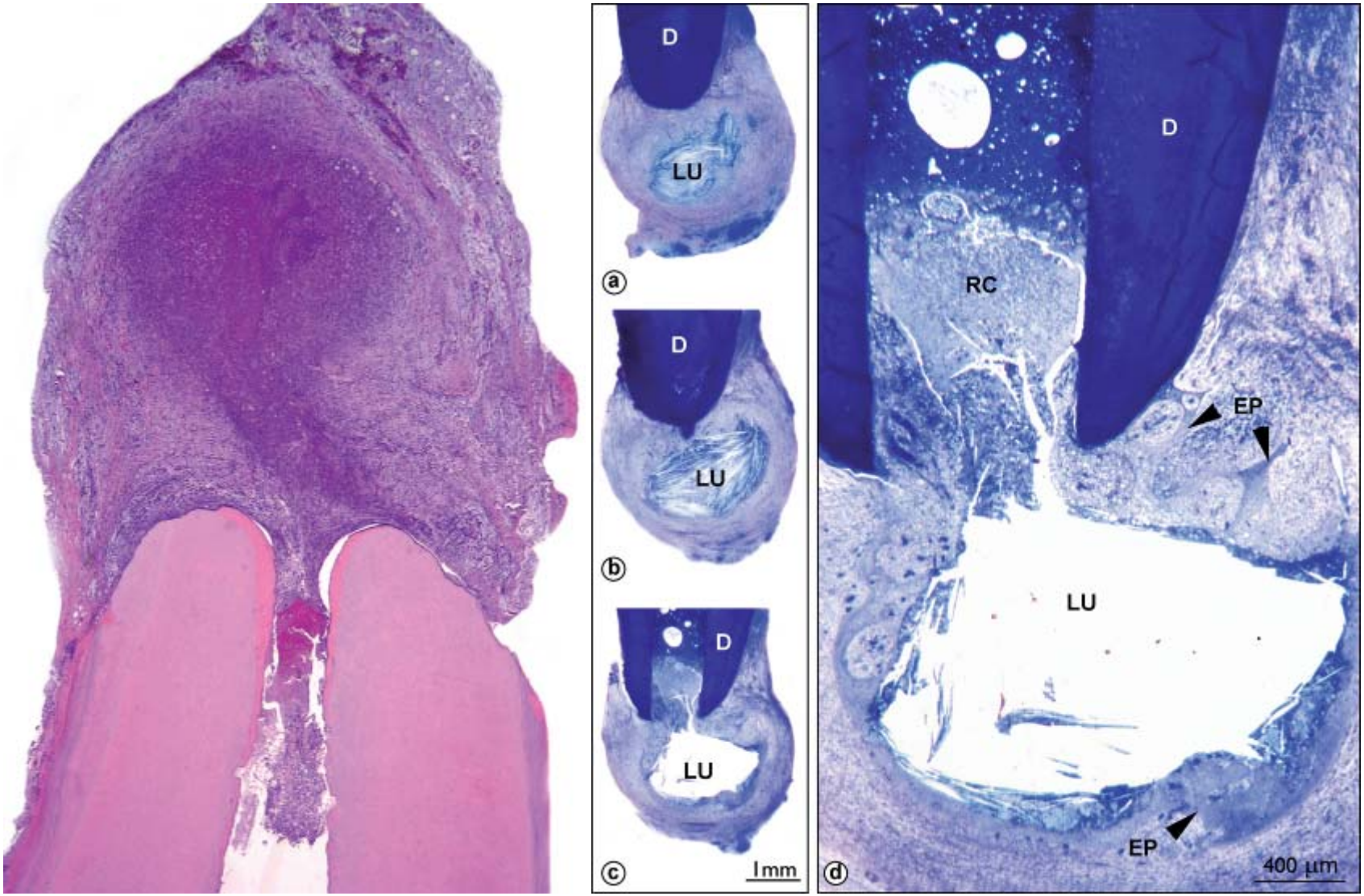

(4)
(2) OSTEOCLAST
(1)
(2)
OSTEOBLAST
• differentiation
• activation
• fusion
• survival
(3)
(2) DENDRITIC CELL
T CELL
• survival
• immunostimulatory activity
(1)
(2)
OPG
RANKL
RANK
TRAIL
TACE

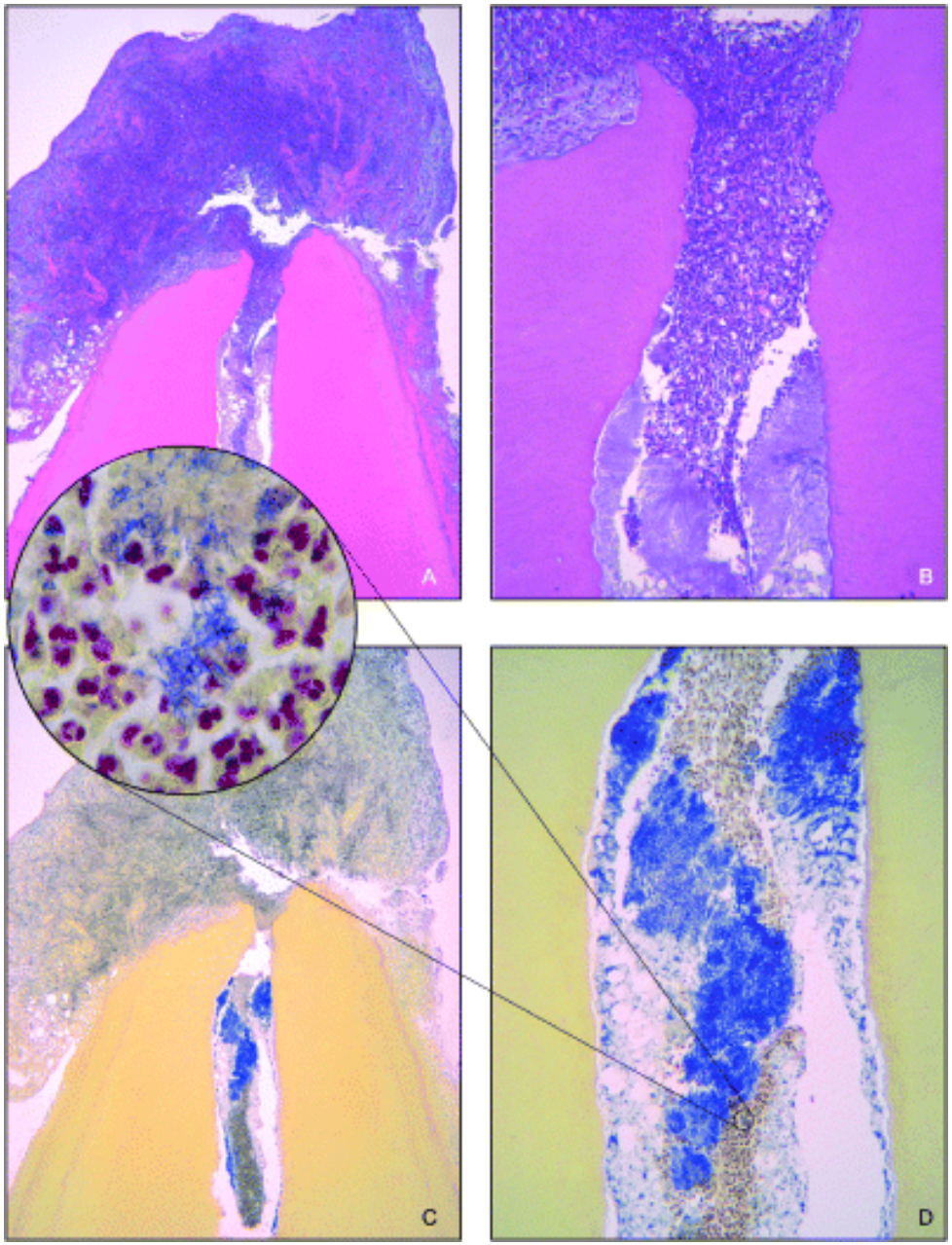


Menezes R, Garlet TP, Letra A, Bramante CM, Campanelli AP, Figueira Rde C, Sogayar MC, Granjeiro JM, Garlet GP. Differential patterns of receptor activator of nuclear factor kappa B ligand/osteoprotegerin expression in human periapical granulomas: possible association with progressive or stable nature of the lesions.
J Endod. 2008 Aug;34(8):932-8

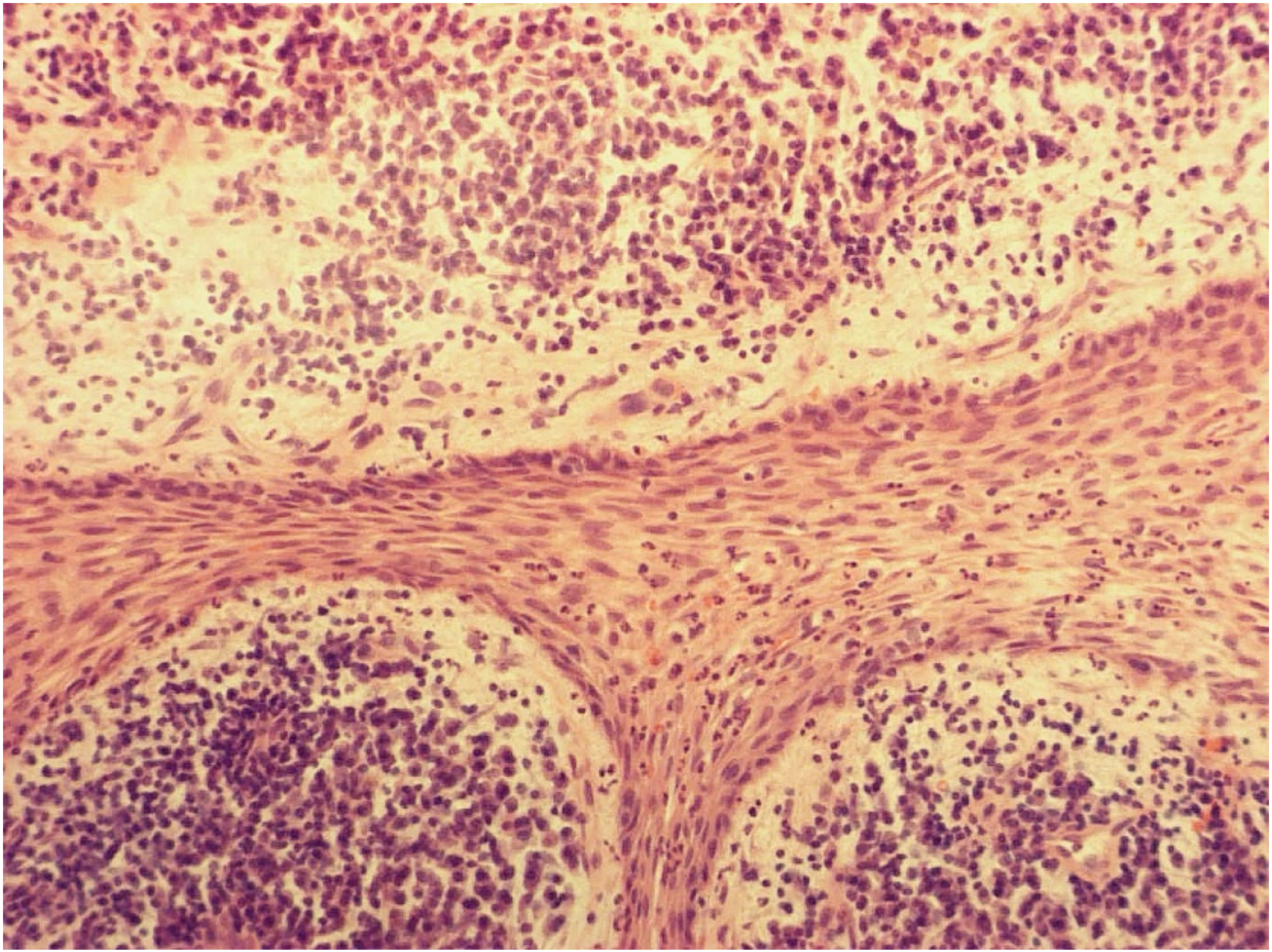


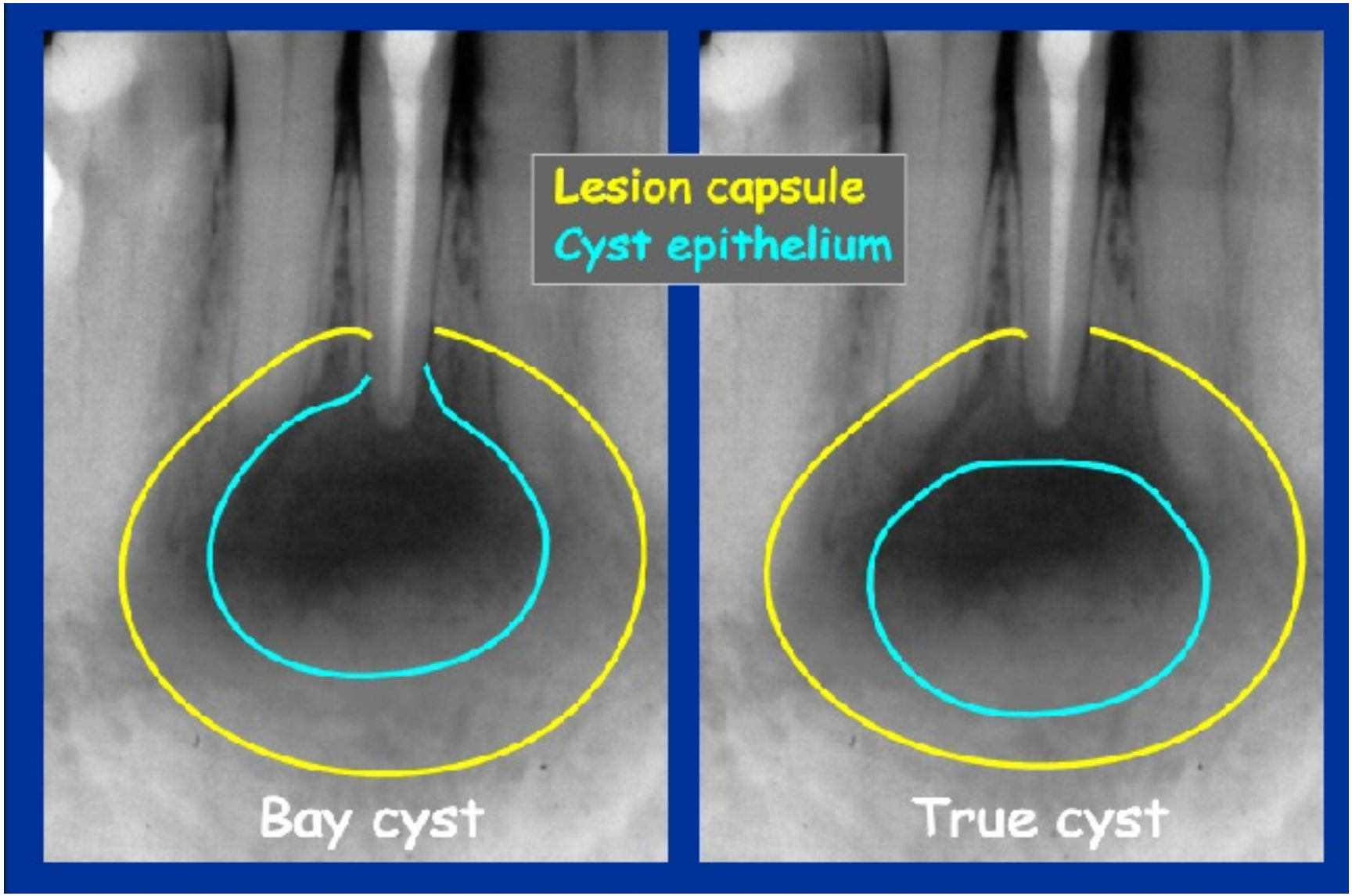
http://en.wikipedia.org/wiki/Osteoclast; 2007-06-21


D
LU
a
D
LU
b
D
LU
c
1mm
D
RC
EP
LU
EP
d
400 µm



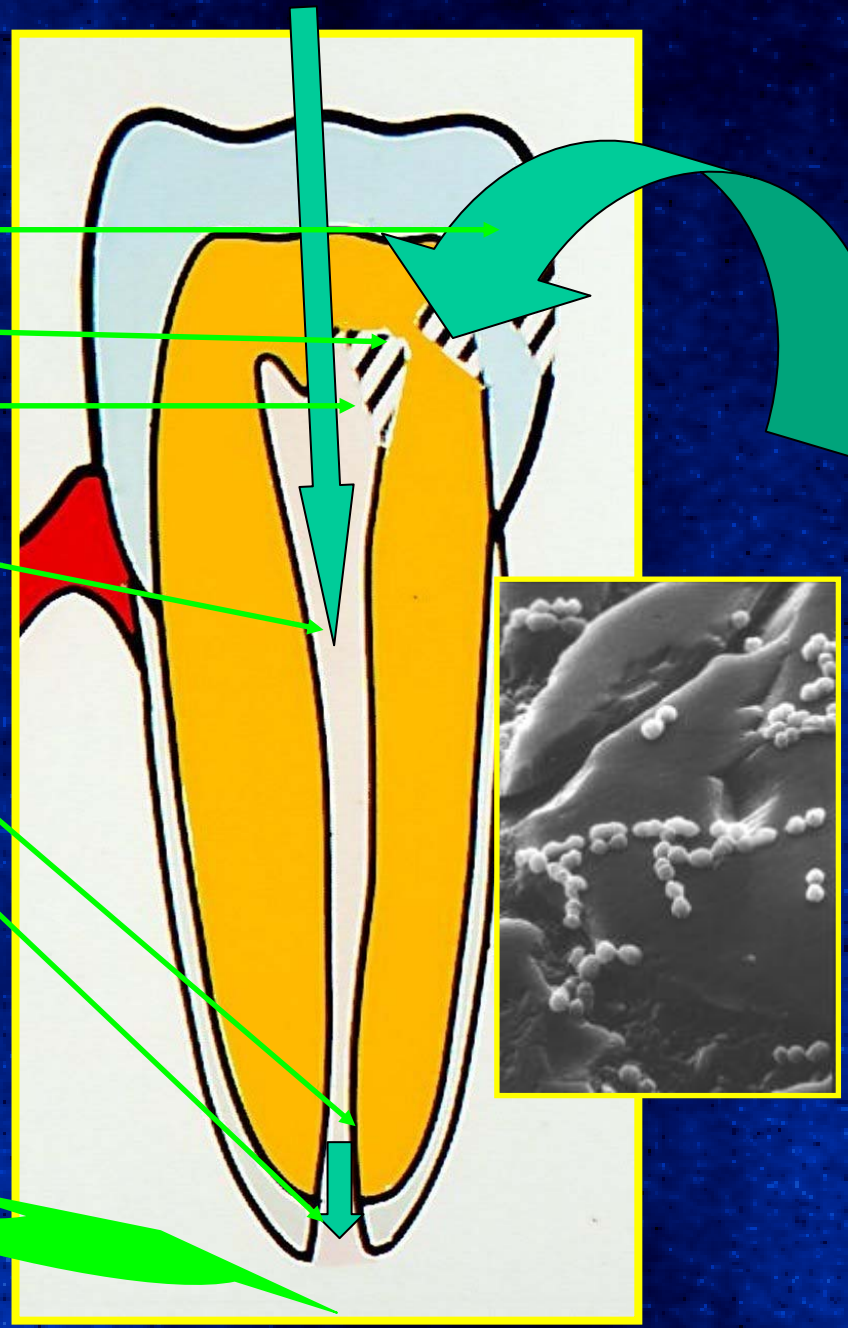
Ricucci
&
Bergenholtz
2004

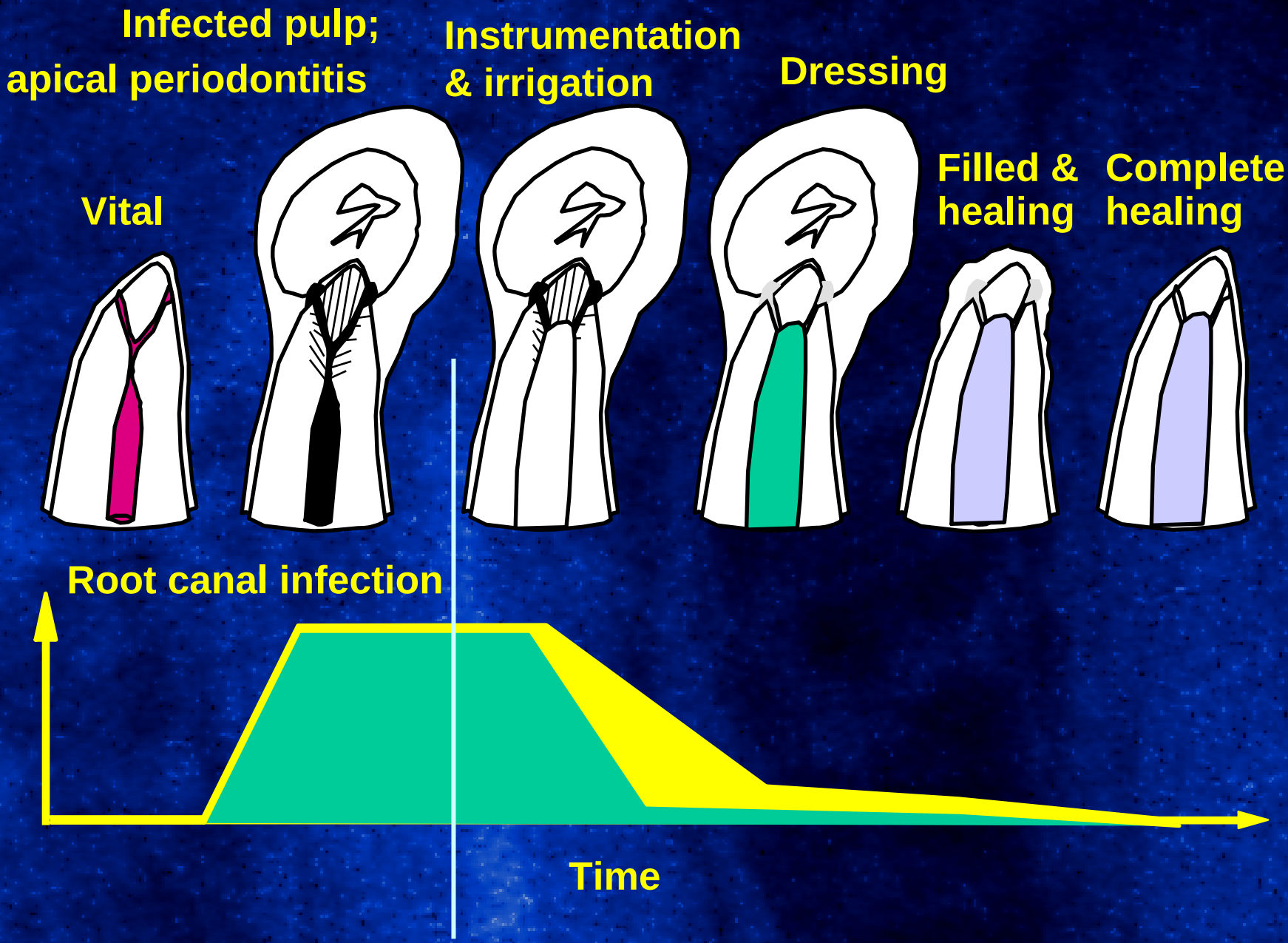



Lesion capsule
Cyst epithelium
Bay cyst
True cyst

- Dentin protection
- Pulp capping
- Partial pulpotomy
- Pulpotomy
- Pulpectomy
- Disinfection

- Pain control
- Antibiotics




Infected pulp;
apical periodontitis
Instrumentation
& irrigation
Dressing
Vital
Filled &
healing
Complete
healing
Root canal infection
Time

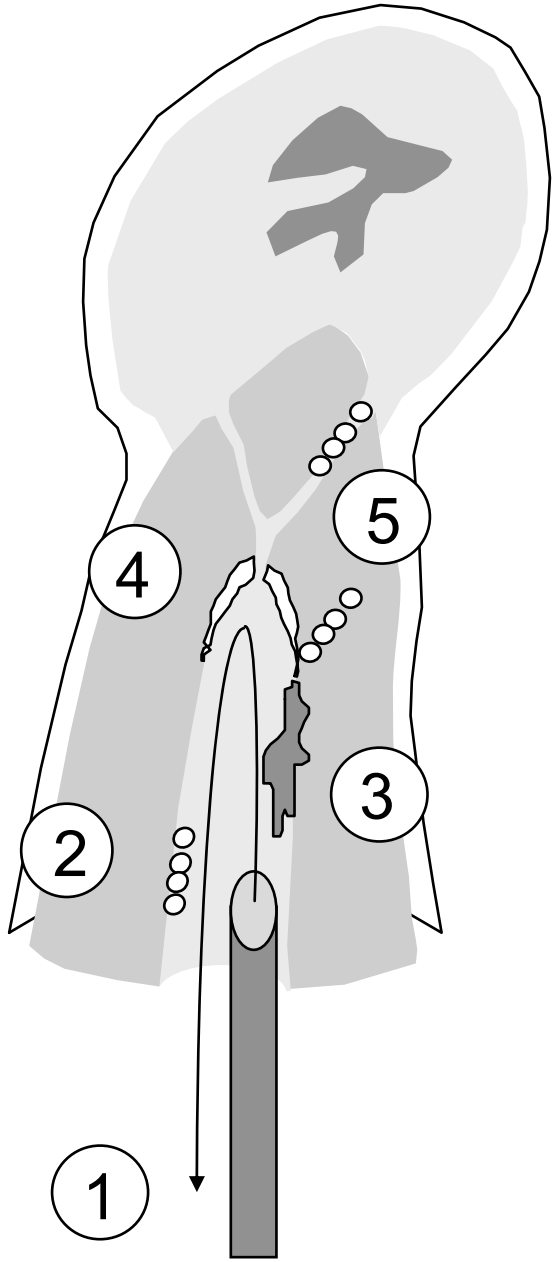
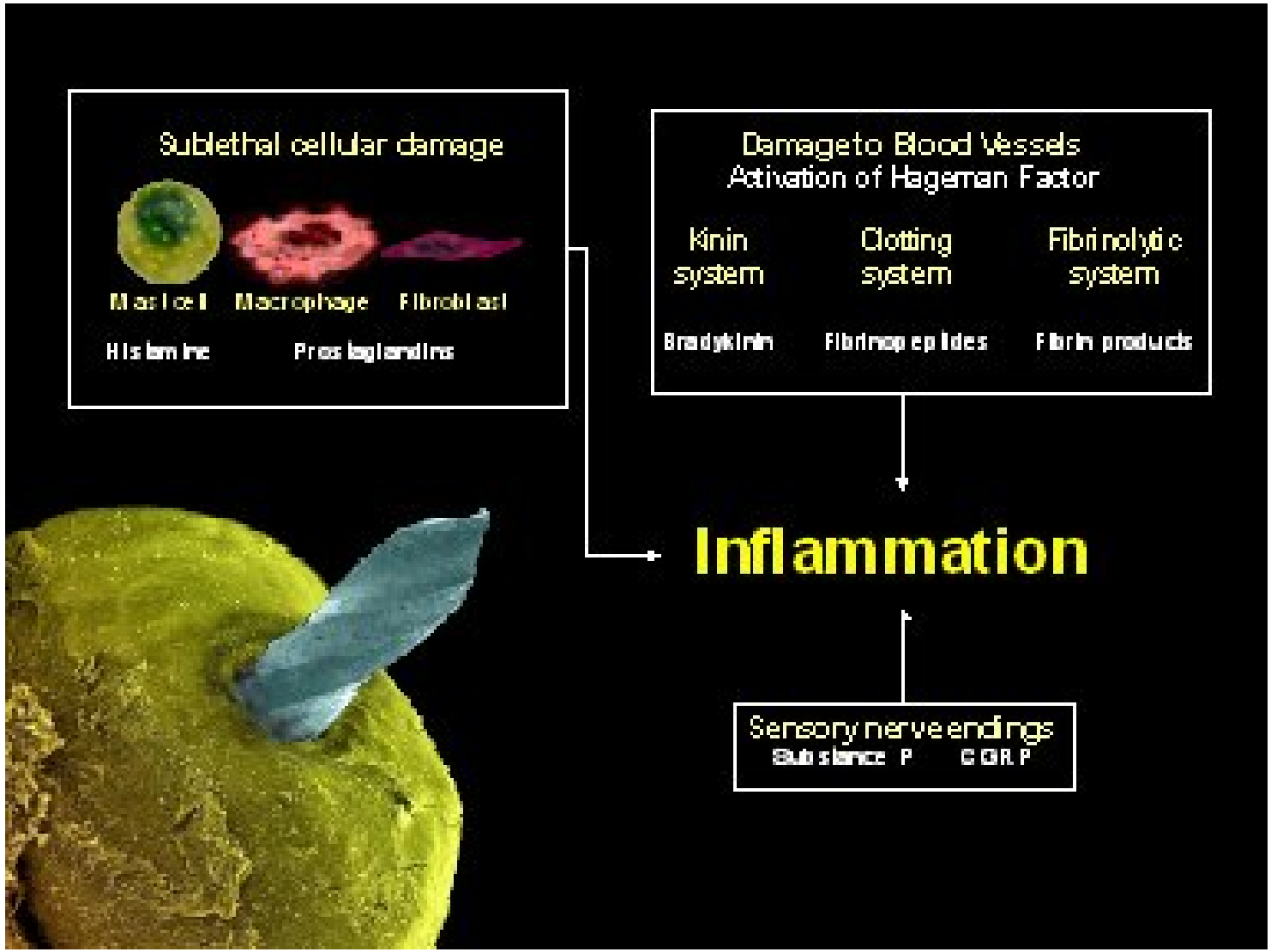
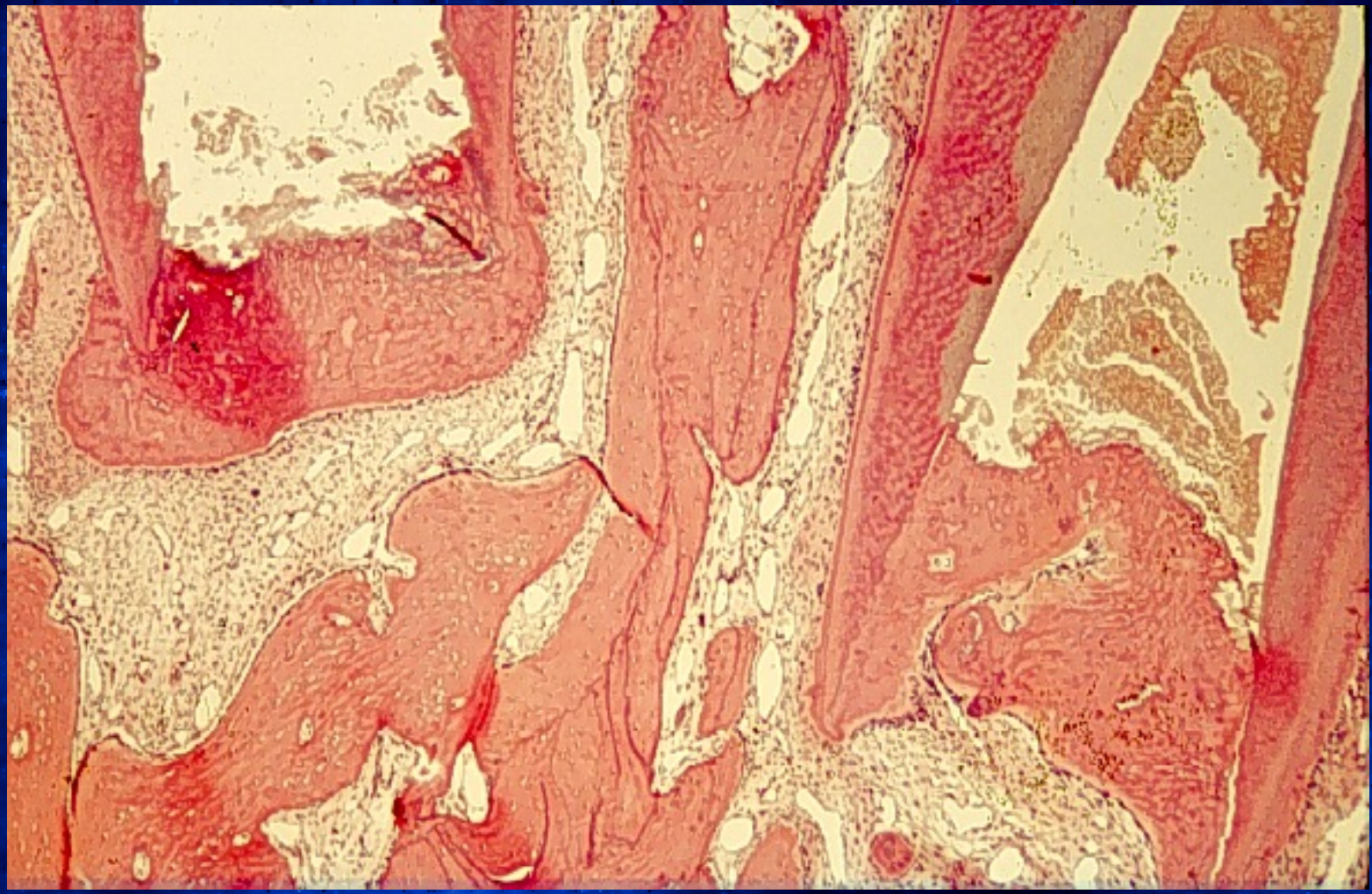
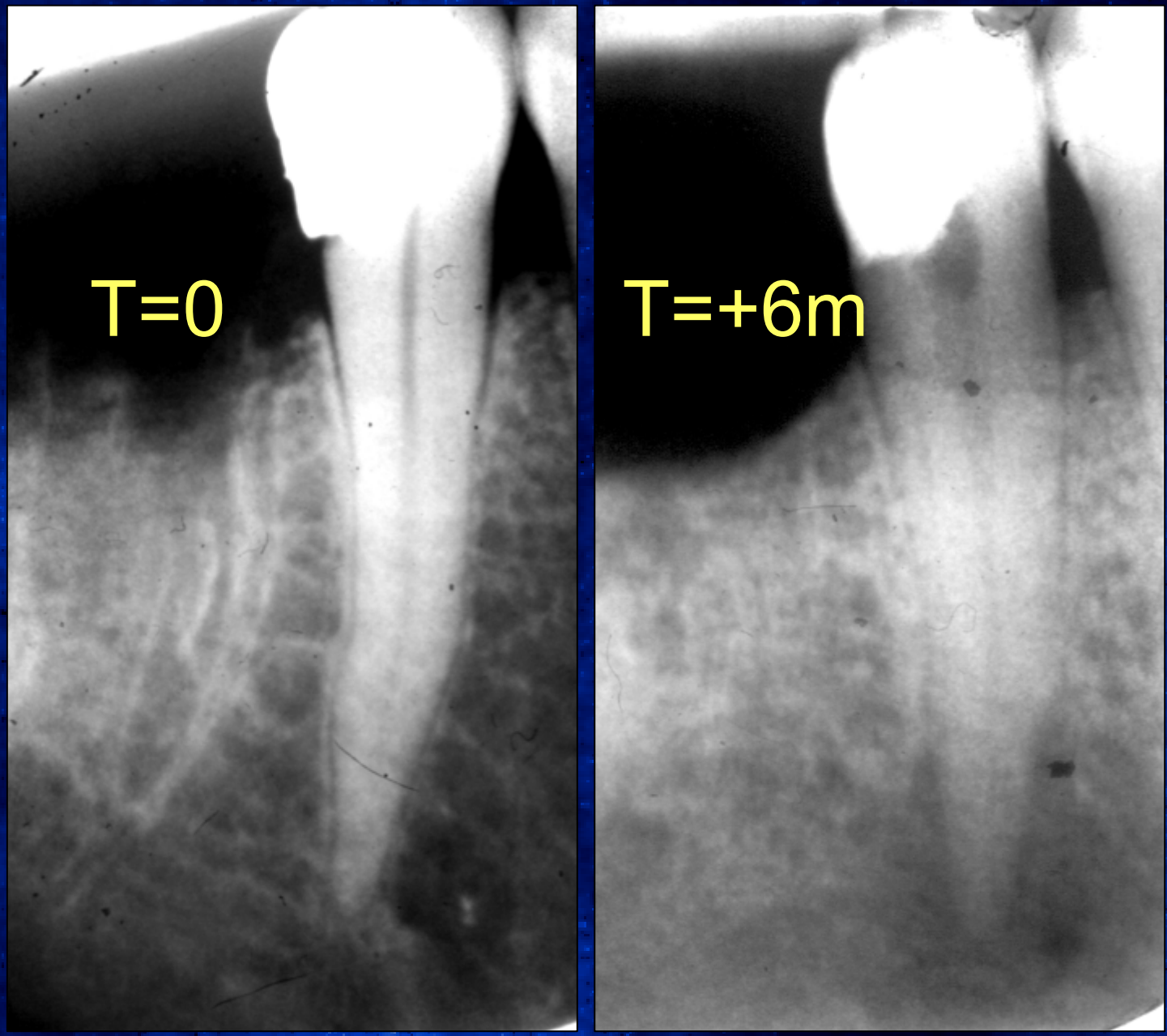


Fig. 11.3

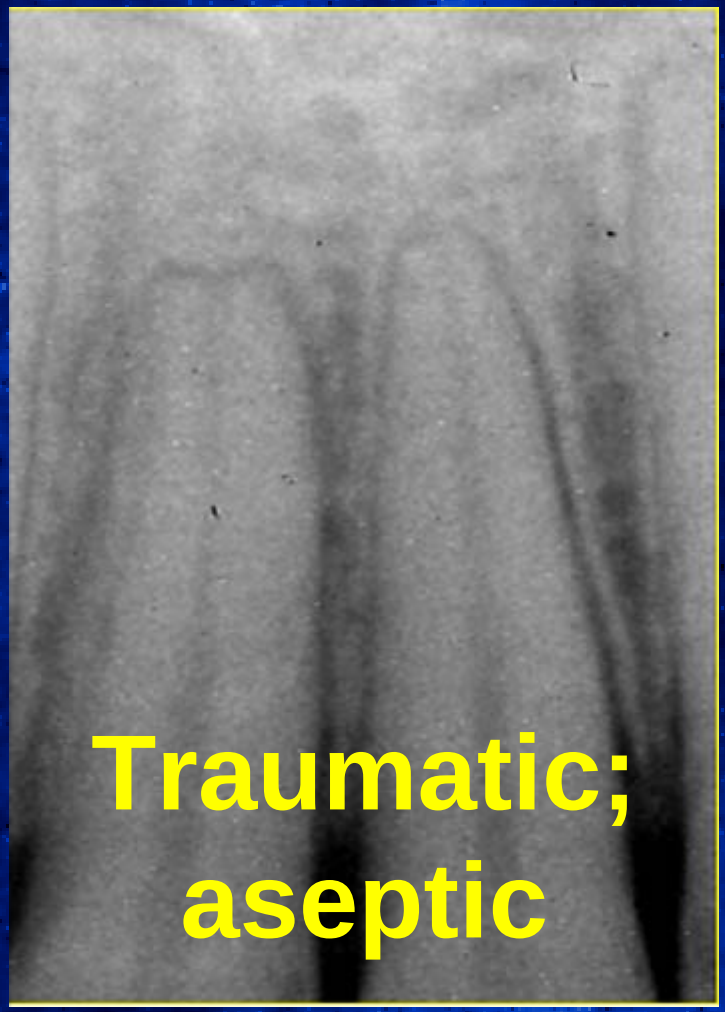


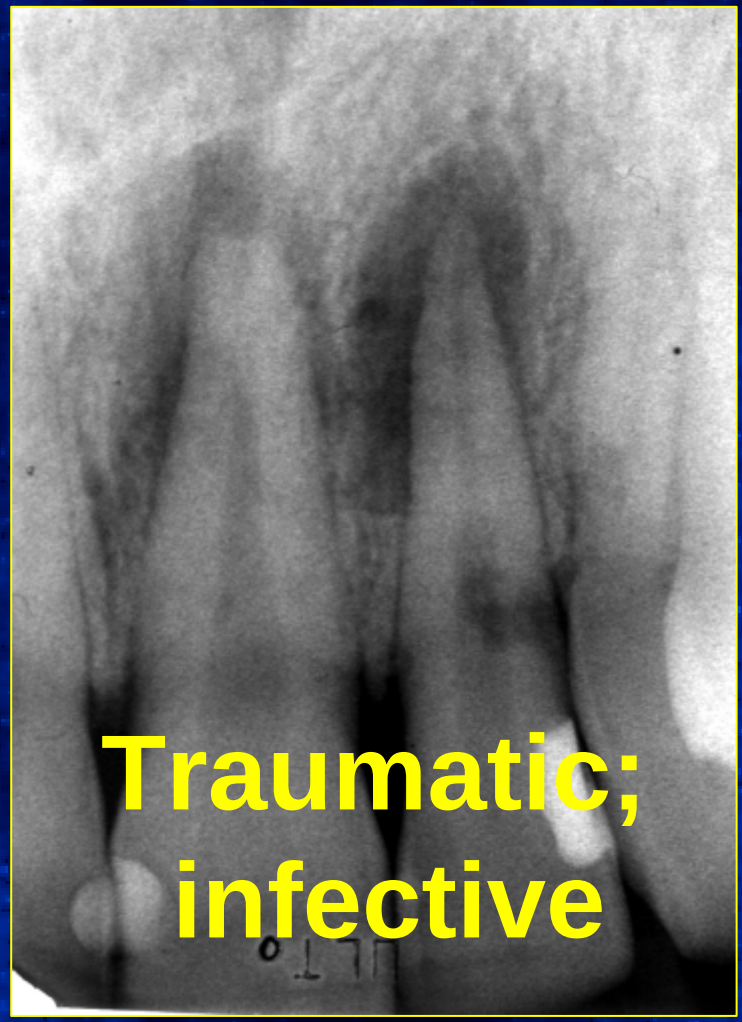
Siqueira & Barnett 2004




T=0
T=+6m

Apical root resorption





Side diagnoses: Vertical fracture