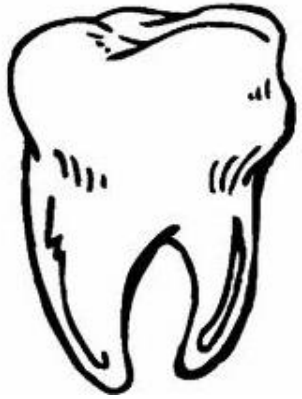
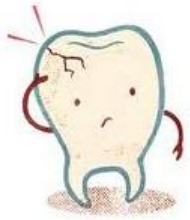
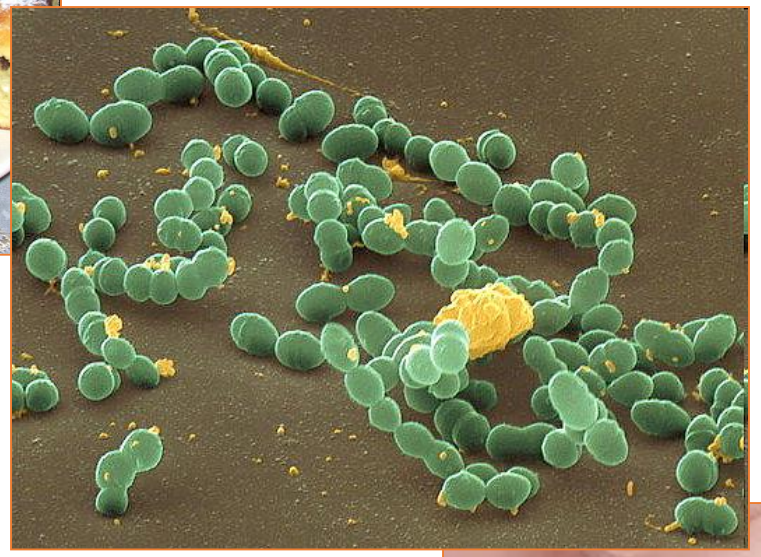


Caries pathology

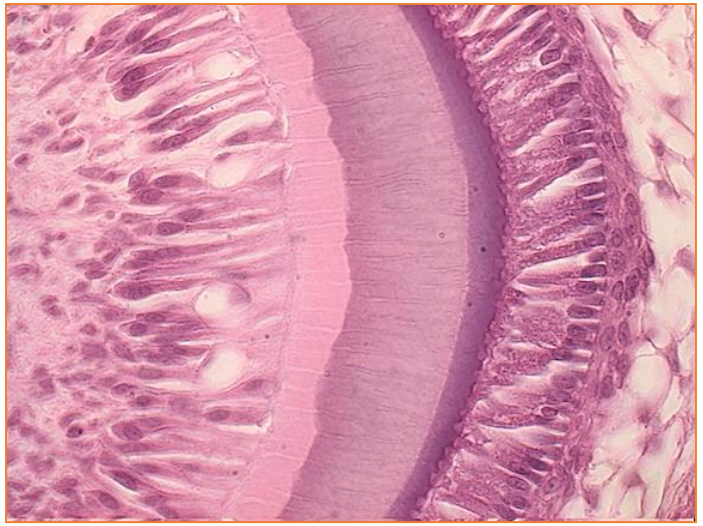
Attila Zalatnai



Contributors



Ameloblasts



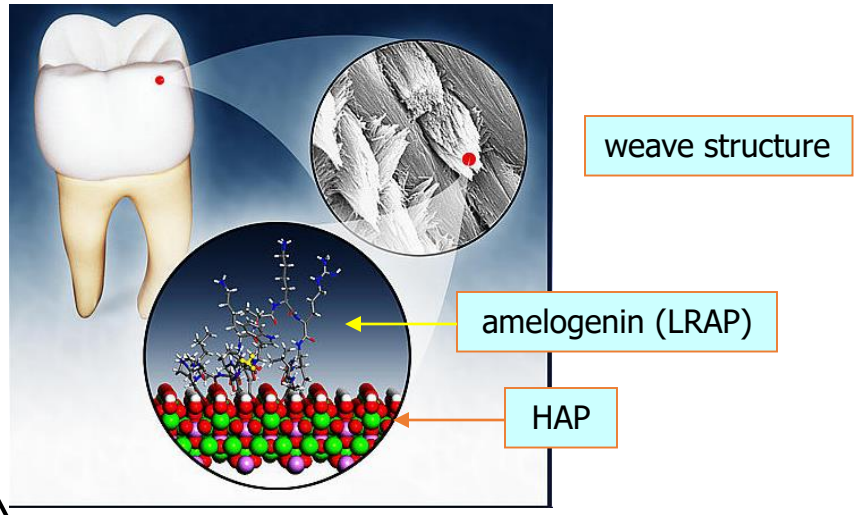
Minerals

90 % hydroxyapatite

$Ca_{10}(PO_4)_6(OH)_2$

3 % fluoroapatite

+ carbonates, silicates, other metals



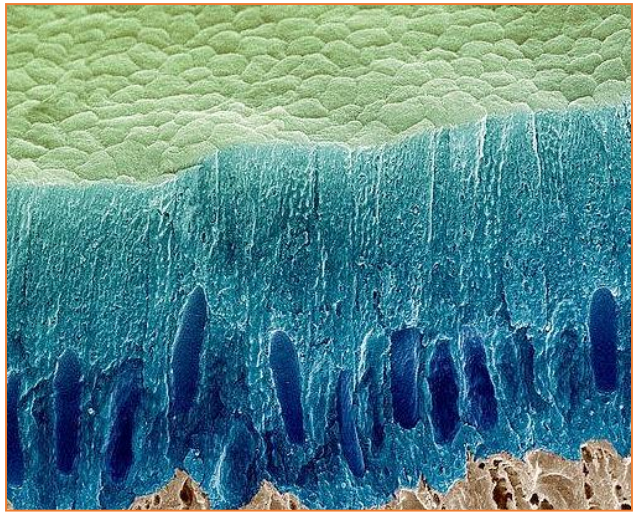
Proteins

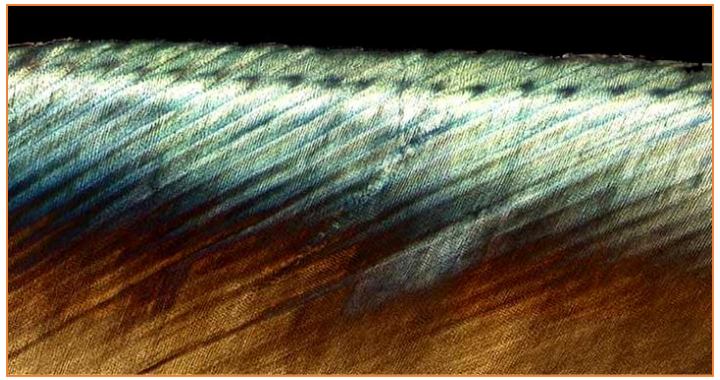
90 % amelogenin

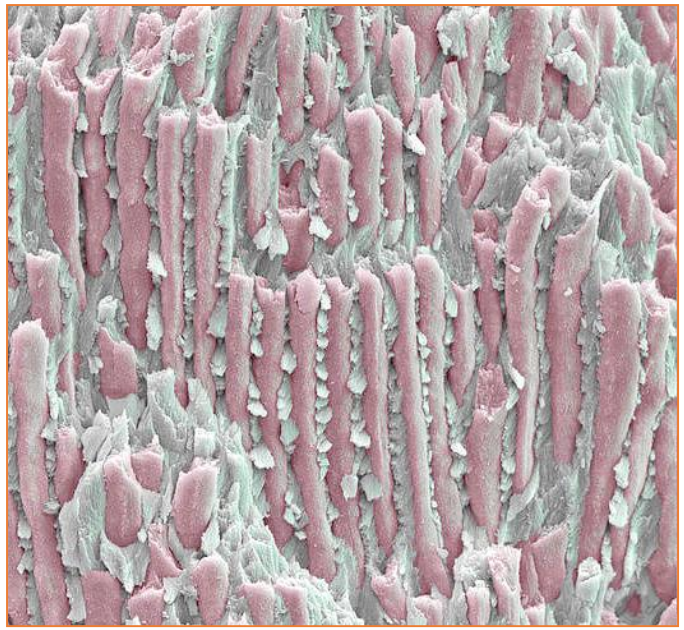
5 % ameloblastin, enamelin, amelatin, apin, MMP20

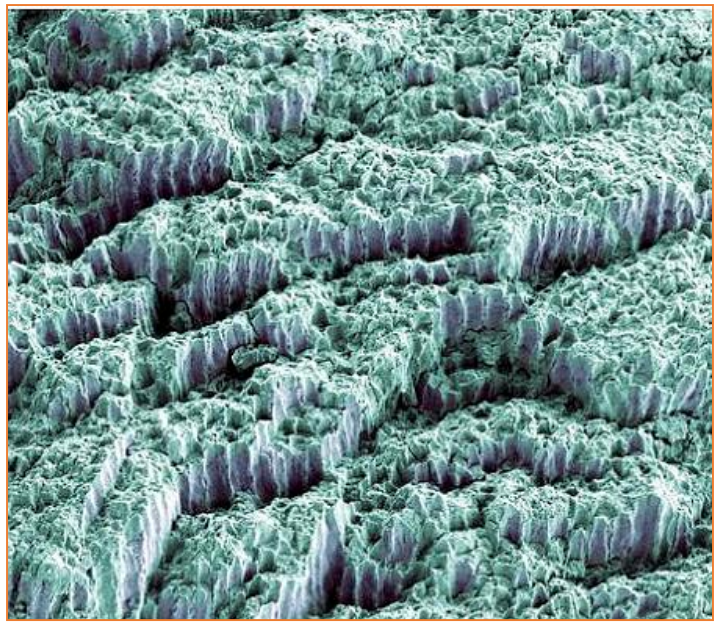
COLLAGEN Ø !

Healthy enamel









Enamel: hardest biological tissue!

Mohs scale (= scratch resistance of various minerals):

1 - talc

2 - gypsum

3 - calcite

4 - fluorite

5 - apatite

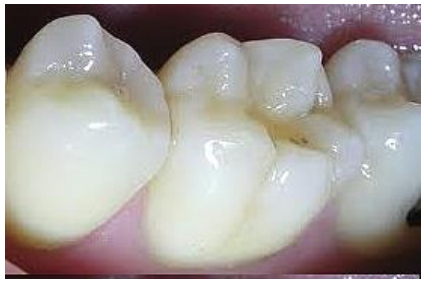
6 - feldspar

7 - quartz

8 - topaz

9 - corundum

10 - diamond



Key contributors: Streptococcus mutans, Str. Intermedius,
(Lactobacillus acidophilus, Actinomyces viscosus)

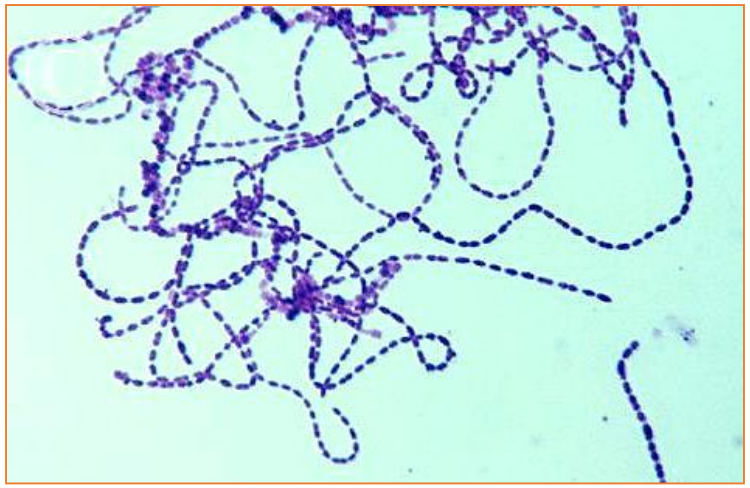
Oral cavity: 25 Streptococcus-species (20% of bacterial flora)

S. mutans: Gram-positive, facultative anaerobic bacterium

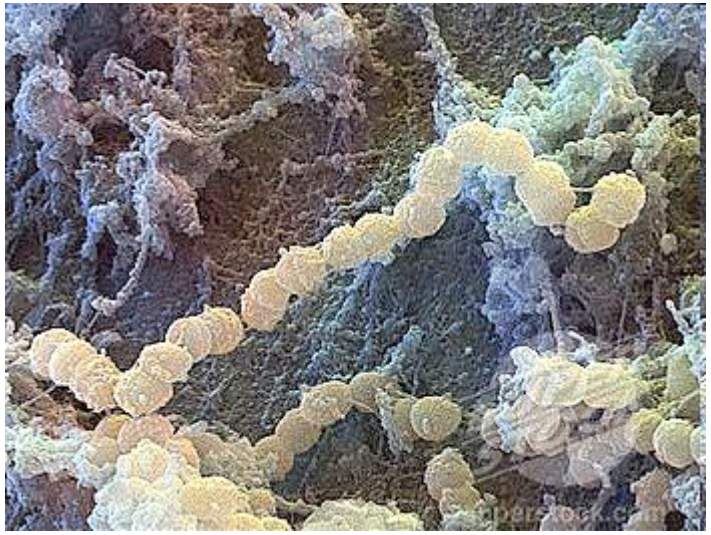
(already present in 2-6 years old children)

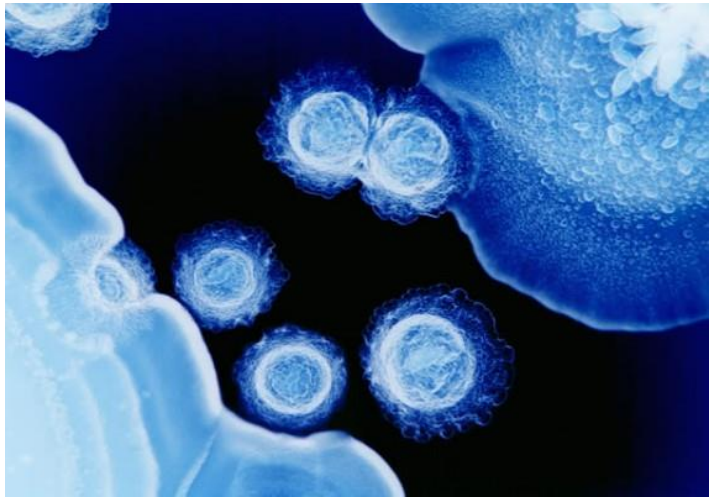
Serotypes: **c**, (70%), e, f, k

k - high affinity to the endocardium (collagen-binding protein /CNM/ content!)



Key contributors: Streptococcus mutans, Strcc.intermedius,
(Lactobacillus acidophilus, Actinomyces viscosus)





For their accumulation sucrose and synthesized enzymes (glucosyltransferases) necessary

(only these Strcc.-i possess this enzyme!)

- Adherence with glycoproteins of dental pellicle (adhesin, etc.)

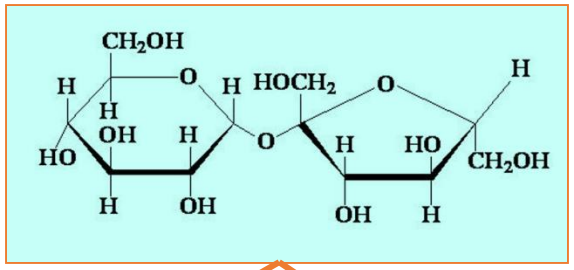
-Bacterial accumulation in the presence of sucrose, extracellular glucan production from glucose (polysaccharide)

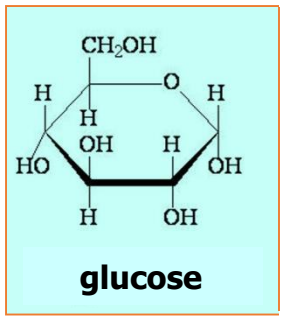
- Bacterial aggregation by their glucan-binding receptors

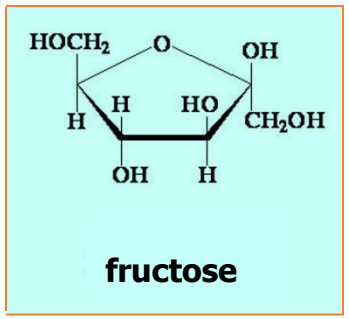
- Acid production by the bacteria

(lactic acid, formic acid, acetic acid, propionic acid)

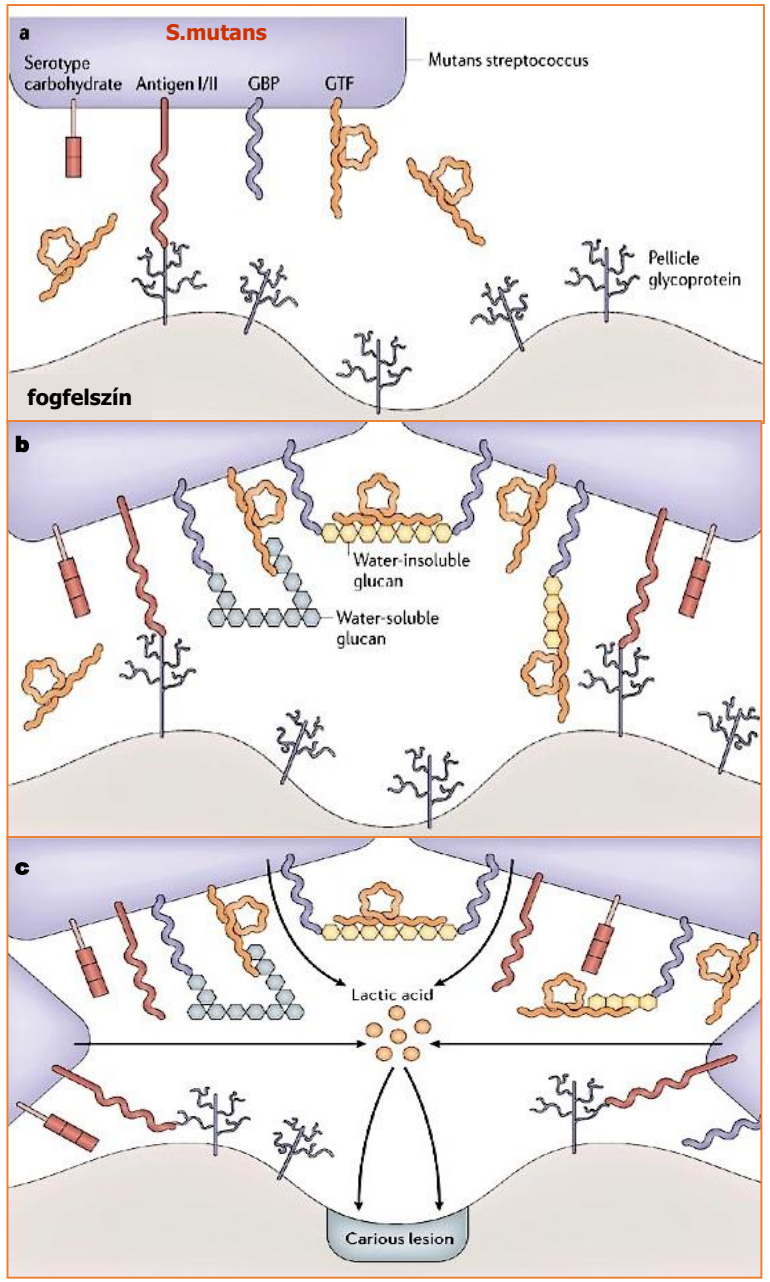
Sucrose (saccharose)







(necessary for the glycolysis)



attachment

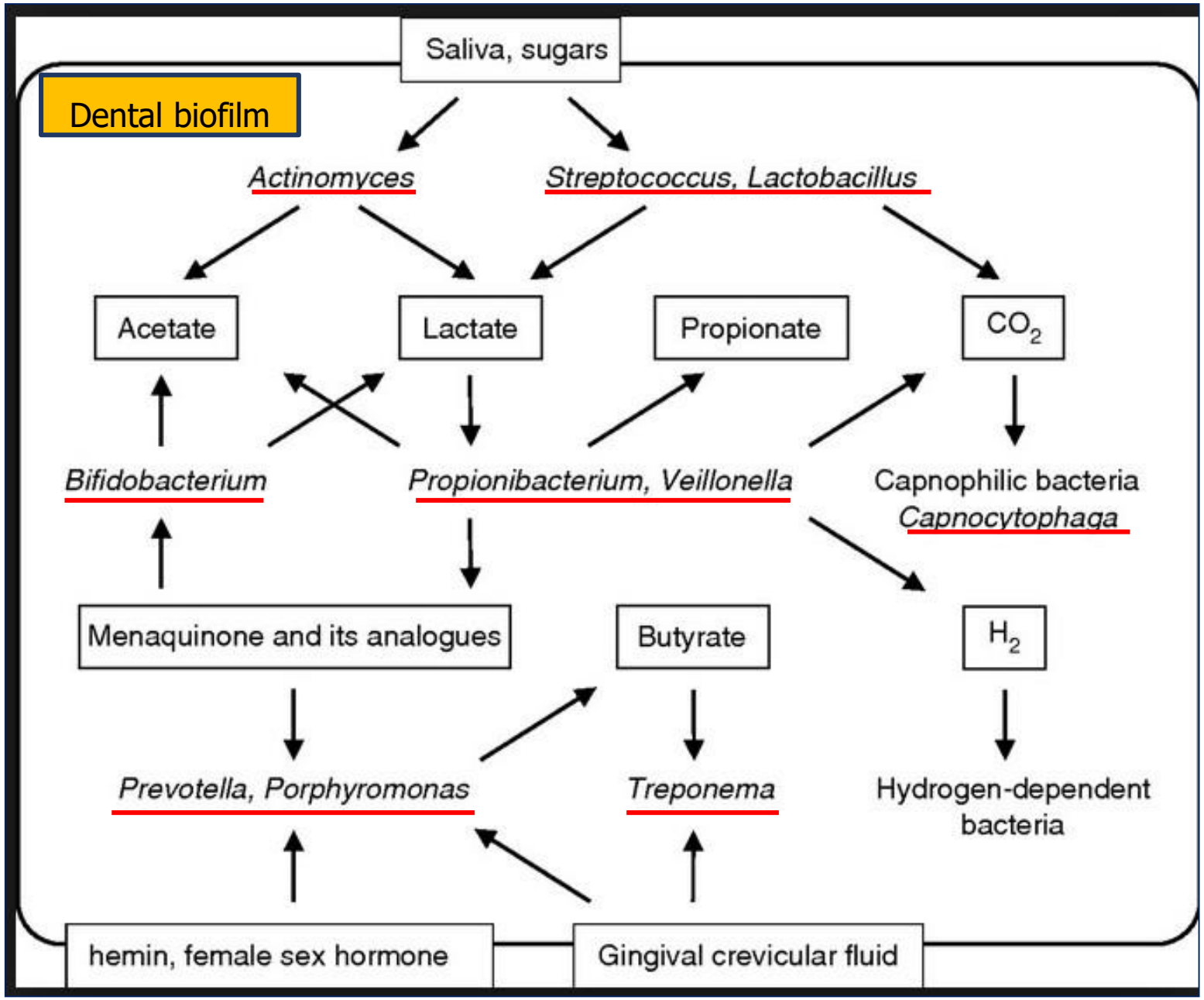
↓

aggregation,

glucan production

↓

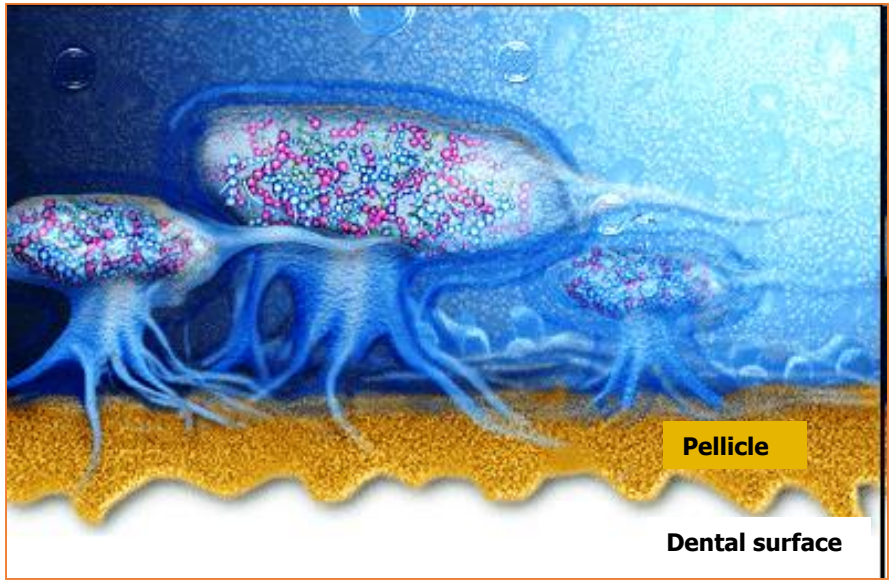
acid production


Dental biofilm
Saliva, sugars
Actinomyces
Streptococcus, Lactobacillus
Acetate
Lactate
Propionate
CO_2
Bifidobacterium
Propionibacterium, Veillonella
Capnophilic bacteria
Capnocytophaga
Menaquinone and its analogues
Butyrate
H_2
Prevotella, Porphyromonas
Treponema
Hydrogen-dependent bacteria
hemin, female sex hormone
Gingival crevicular fluid

Dental plaque

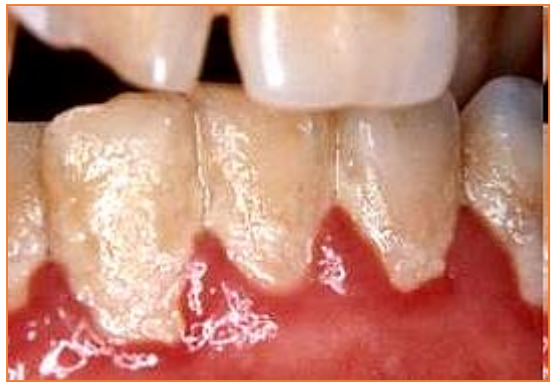
Biofilm

1. Saliva proteins, glycoproteins, some bacterial molecules - fast absorption to the dental surface (pellicle) – primary colonization
2. Specific interactions between the cell surface and the receptors of pellicle (irreverzible)
3. Secondary colonization (new specific interactions)
4. Proliferation of the adherent cells, confluent growth (> 600 microorganisms)
5. Calcification (tartar)



Composition of dental plaque

Not uniform (changing in quantity of saliva, different redox-potentials)



Over 100, various bacteria can be found

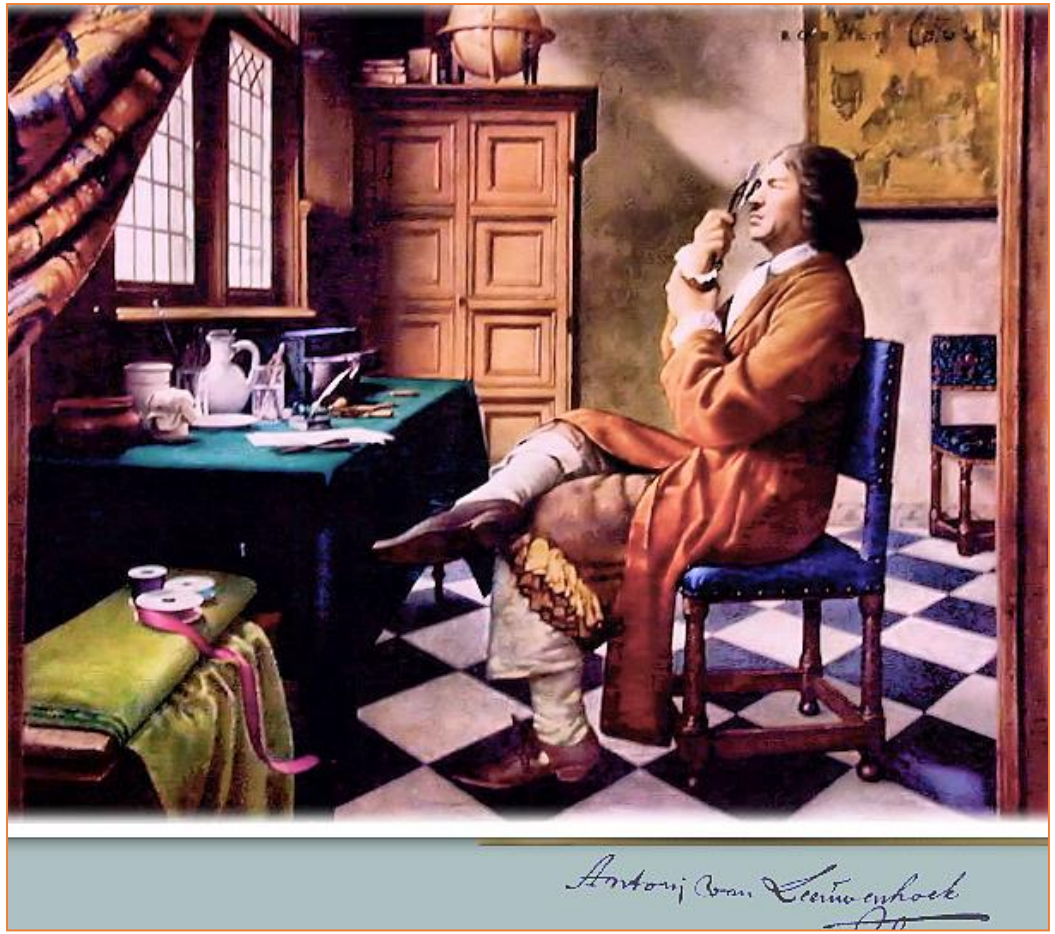
- Streptococci
- Actinomyces species
- Anaerobic Gram-positive rods
- Neisseria
- Veillonella
- Anaerobic Gram-negative rods

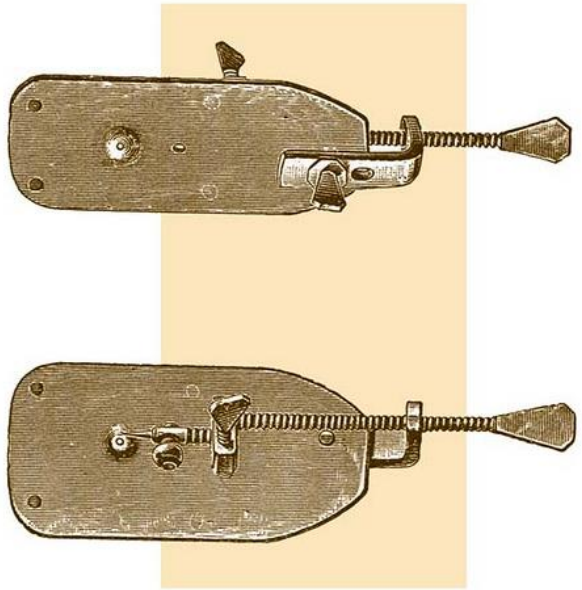
+ cellular debris, proteins, lipids, ions

Early: parallel orientation to the surface,

After 70-10 days: irregularly structured

A.v.Leeuwenhoek (1680)





Other risk factors

- Decreased secretion of saliva (decreased buffering, xerostomia)
- Teeth malalignment
- Medicinal drugs (antihistamines, antidepressants)
- Smoking
- Vitamin-D-deficient conditions
- GERD/erosion: risk in adults, but not in children
- Genetic background

MULTIFACTORIAL

Demineralization

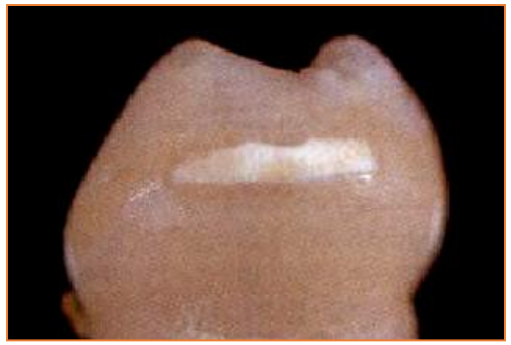
Acids enter the enamel

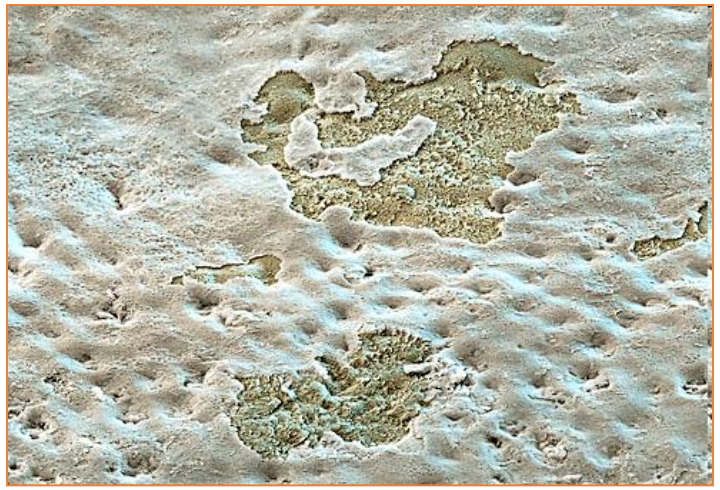
Critical value: below pH 5,5

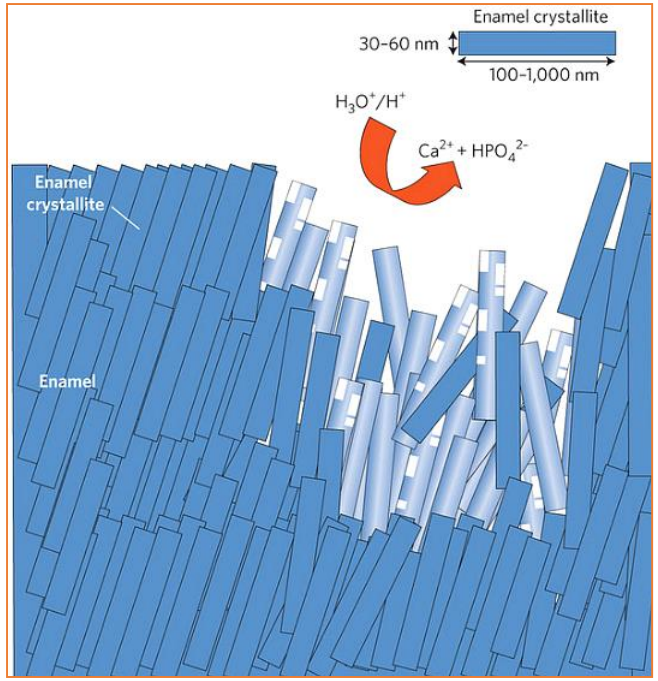
Enamel dissolution is related to the H^+-concentration

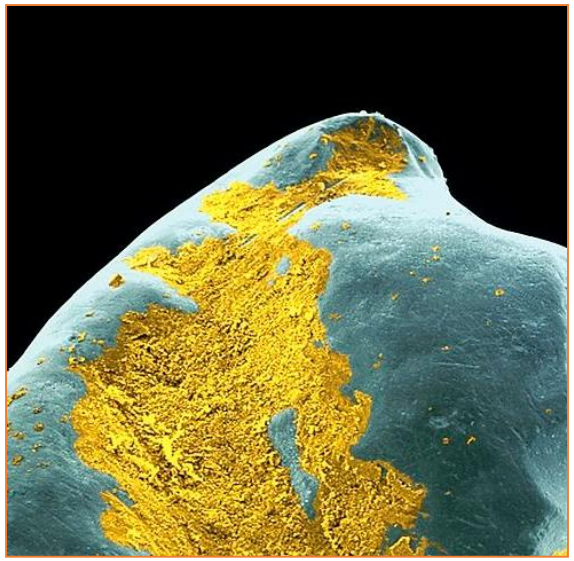
Ca, P release

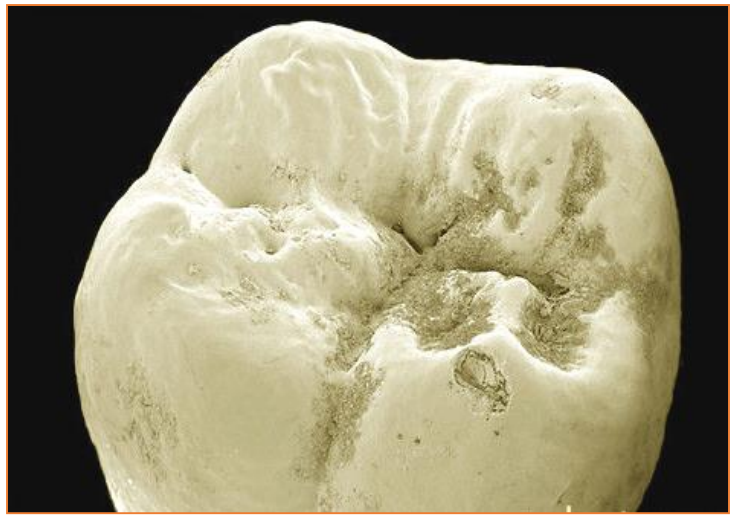
1. Early change: soft, whitish-opaque area, porous surface






Enamel crystallite
30–60 nm
100–1,000 nm
H_3O^+/H^+
$Ca^{2+} + HPO_4^{2-}$
Enamel crystallite
Enamel





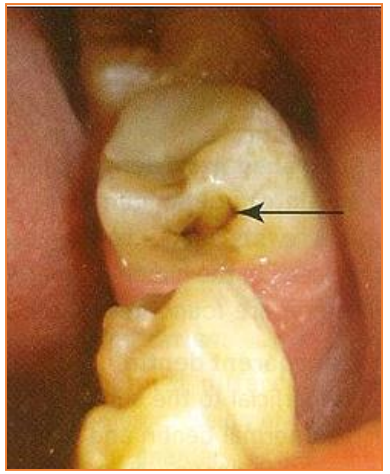
Caries

2. Arrested stage

Can be reversible at the beginning

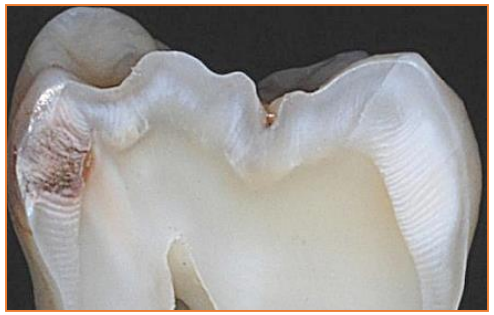
(Ca, P, F influx – remineralization)

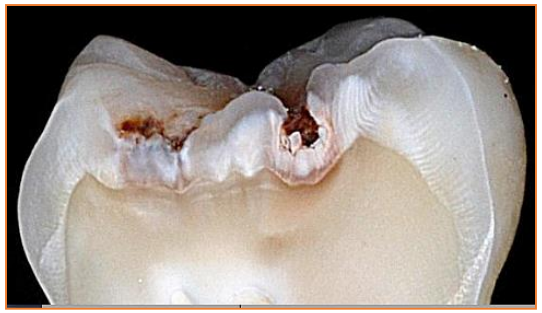
Resistant surface!



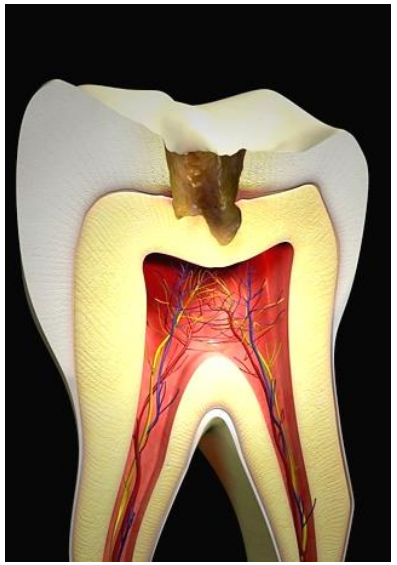
3. Superficial stage

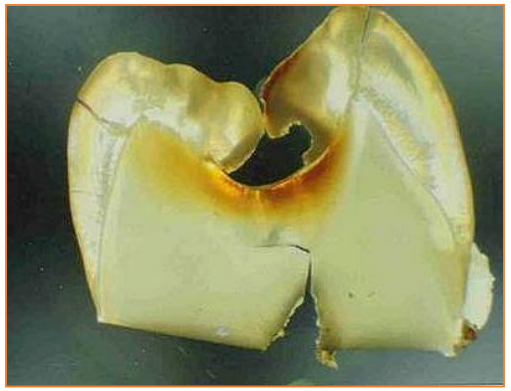
(only enamel is involved)

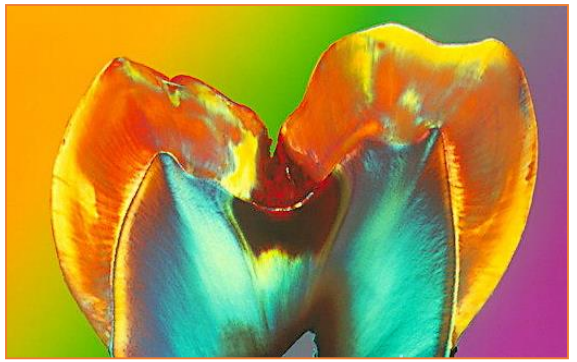




4. Dental caries

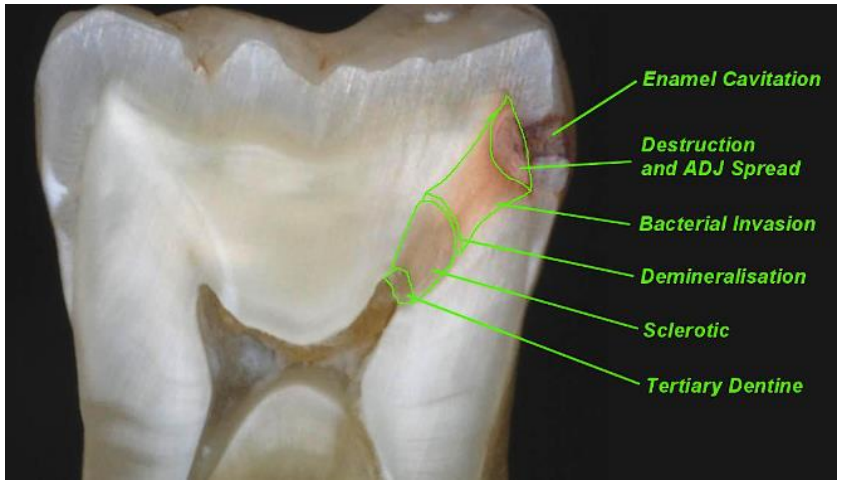






After reaching the dentin the process progresses fast

Odontoblasts proliferate → reactive dentin formation



Enamel destruction

Necrotic, liquefied dentin, transversal fissures

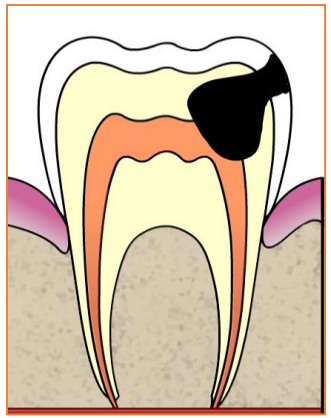
Bacterial invasion along the tubules, proteolysis

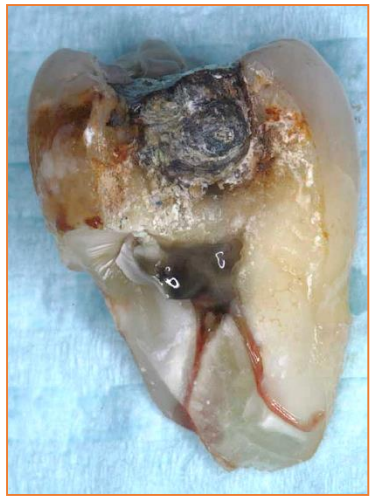
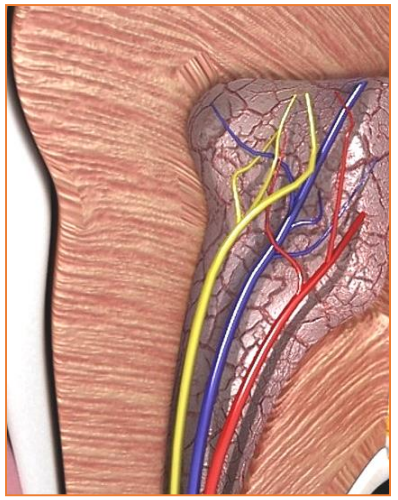
Acid production (no bacteria et this region)

Odontoblastic calcification inside the tubules

Tertiary dentin formation

5. Profound caries (open pulp)

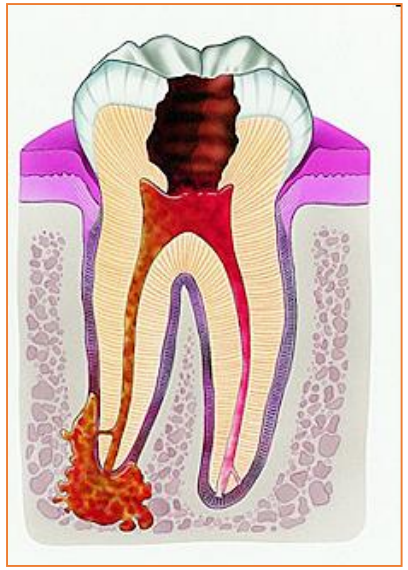


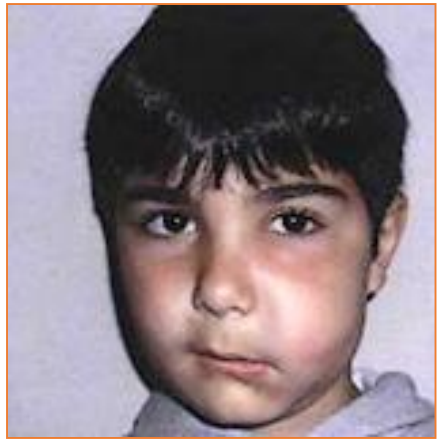
Pulpitis
- Acute (mainly children, adolescents)
  - serous, purulent
  - gangrenous
- Chronic (hyperplastic; „pulpa-polyp”

- reversible
- irreversible

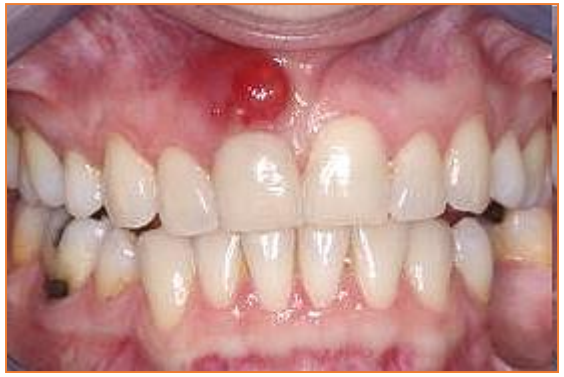
Apical periodontitis

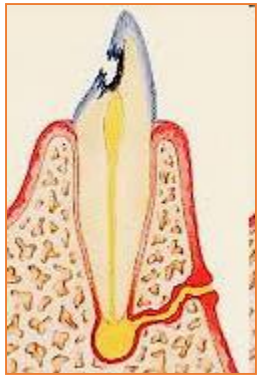


- Symptomless periodontitis
- Symptomatic periodontitis
- Acute periapical abscess
- Chronic periapical abscess
- Periostitis
- Osteomyelitis
- Phlegmone



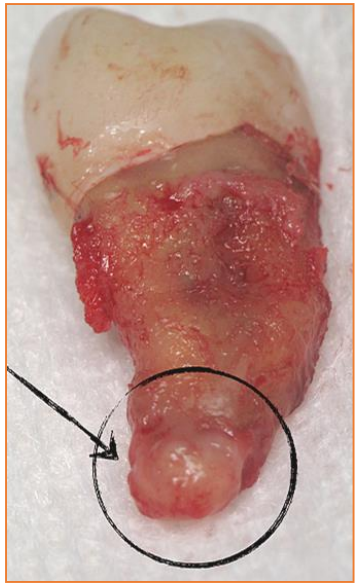
Periapical abscess



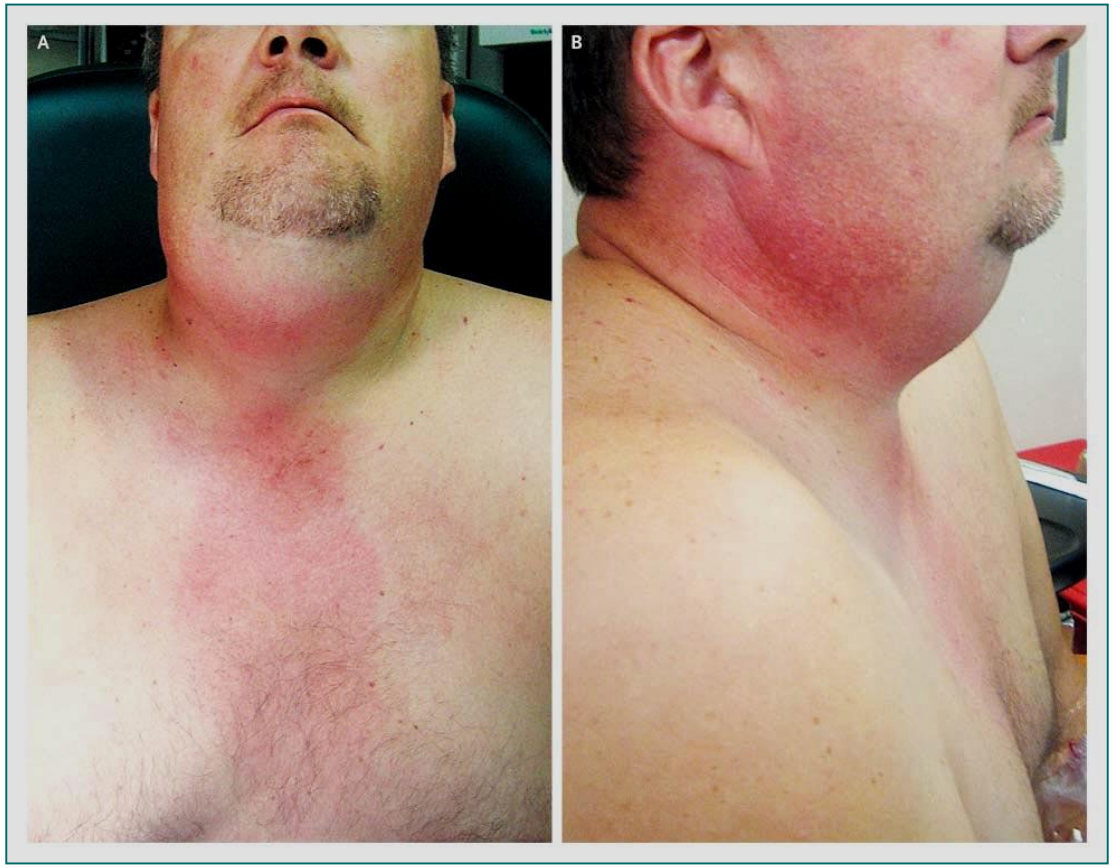


Consequences:

- fistule
- Ludwig-angina (floor of mouth, neck)
- cavernous sinus thrombosis
- periapical granuloma
- periapical cyst (radicular cyst)



Ludwig-angina



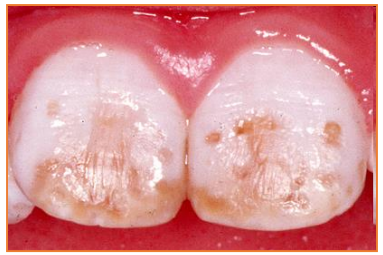
Regressive teeth alterations

Enamel hypoplasia: decreased activity of ameloblasts/ ameloblast degeneration

congenital or acquired (Vitamin-D deficiency, celiac disease!)

uneven attrition

Chronic fluoride poisoning (dental fluorosis):

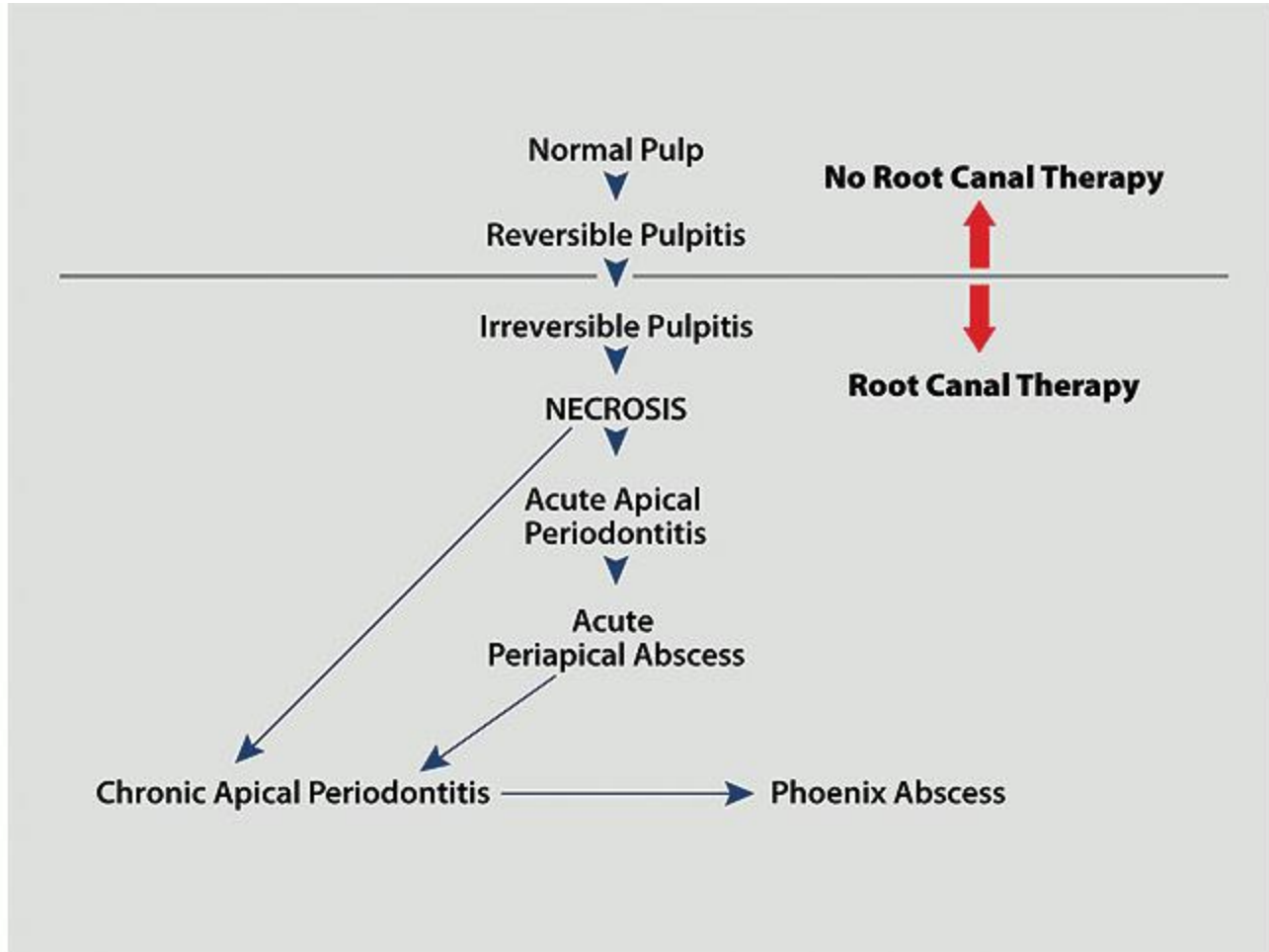


- significantly increased fluoride ingestion during tooth development (> 2 ppm ib drinking water)
- increased amelogenin-concentration, delayed removal
- degeneration of ameloblasts, odontoblasts
- decreased enamel mineralization
- mottling! brown discoloration

Acquired odontoporosis: lacunar dentin resorption

Porodontia: spontaneous opening of the root canal

Postchemotherapeutic effects – root shortening, enamel hypoplasia….


Normal Pulp
Reversible Pulpitis
Irreversible Pulpitis
NECROSIS
Acute Apical Periodontitis
Acute Periapical Abscess
Chronic Apical Periodontitis
Phoenix Abscess
No Root Canal Therapy
Root Canal Therapy