EARLY THEORIES and CURRENT CONCEPT of DENTAL CARIES

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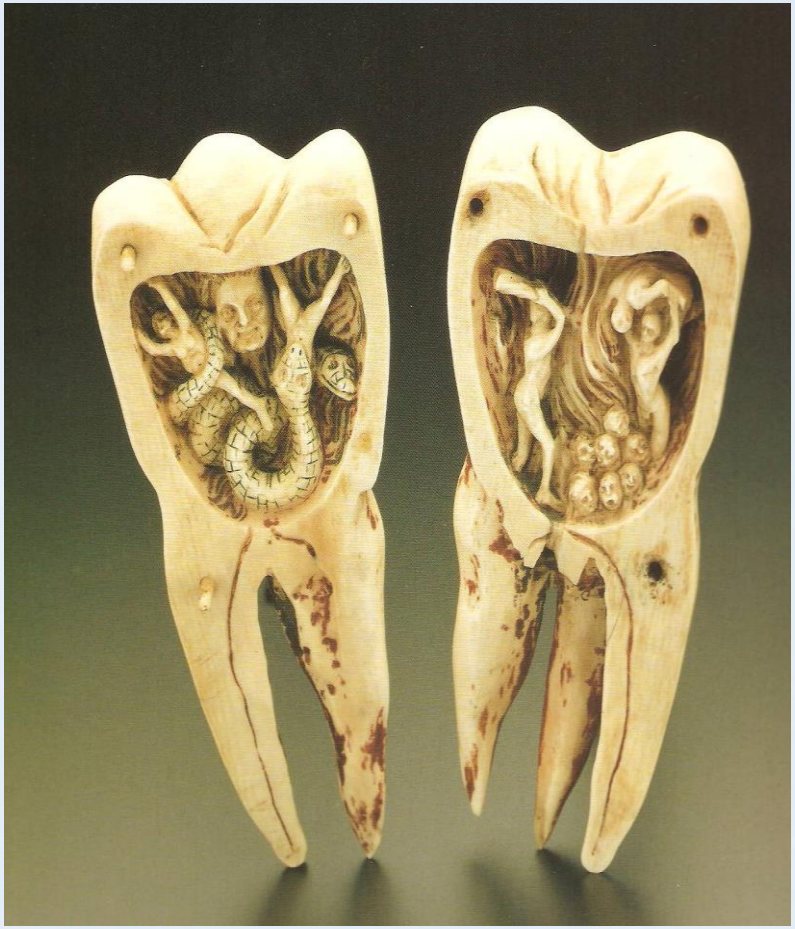


The legend of the Worms

(Deutsches medizinhistorisches Museum Ingolstadt)
Malvin E.Ring: Dentistry

Early theories of Dental Caries

•The legend of the Worms (5.000 BC clay tablet)

Endogenous theories:

- Humoral theory (Hippokrates)
- Vital theory
- Proteolytic theory (Bodecker 1948)
- Phosphate theory (Csernyei 1950)

Exogenous theories:

- Chemical (acid) theory (Parmly and Robertson) 1819
- Parasitic (septic) theory (Erdl and Ficinus) 1843
- Chemico-parasitic theory (1889 MILLER)
- Proteolytic theory (Gottlieb 194?1)
- Proteolysis-chelation theory (Martin-Schatz 1955)
- Sulfatase theory Pincus (proteolytic) (1949)
- Complexing and phosphorila-ting theory (Eggers-Lura 1967)

Endogenous theories:

- Humoral theory The relative proportion of the four elemental fluid of the body determines the person's physical and mental constitution.(blood, phlegm, black bile, yellow bile) (Greek)

- Vital theory :Physiciants of the Middle Ages

  Teeth are the integral part of the body.

  (Celsus, Galen)

- (Hippokrates) systemic and local factors

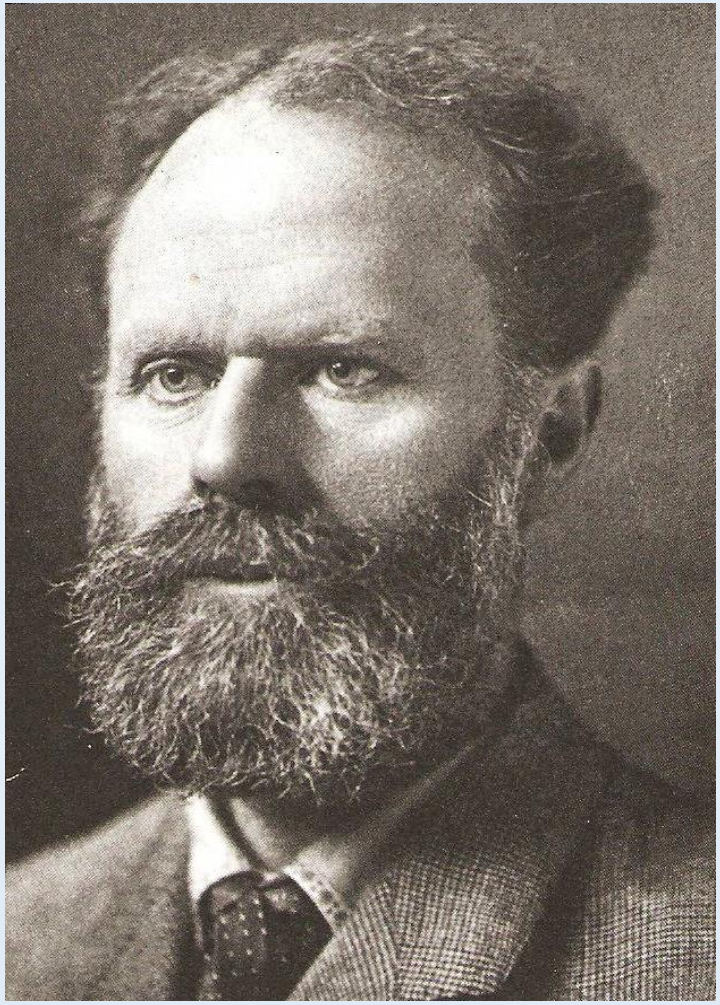
Exogenous theories:

- Chemical (acid) theory: 1819 (Parmly and Robertson)

  Caries starts on the enamel surface, where food putrified and acquired sufficient dissolving power. This dissolving power is inorganic acid (such as sulfuric and nitric). (Ammonia comes from protein, and oxidises) Corrosion.

- Parasitic (septic) theory 1843 (Erdl and Ficinus) „filamentous organism” on the tooth surface. He gave the name of „denticolae”. He (Ficinus) had recognize first the organic matrix in enamel.

Chemo-parasitic theory (1889)
W.D.MILLER



- American scientist
- BA degree in chemistry, physics and mathematics
- In Berlin he met with dr.Abbot
- 1879 DDS degree
- 1884 in Berlin Prof. of operative dentistry
- 1890 Microorganisms of the Human Mouth
- 1907 back to America and he died suddenly (appendicitis)

Exogenous theories:

- **Chemico-parasitic theory** (**1889 MILLER**)

- Koch: bacteriologie
- Pasteur:(fermentation of carbohydrate)
- G.V.Black

Dental caries is a chemo-parasitic process, consisting of two stages: decalcification and dissolution.

-acids are present in the deeper carious lesons

-different microorganisms invade carious dentin

Later Williams (1897) observed the plaque

Exogenous theories:

- Proteolytic theory (Gottlieb 1941)

  He thought, that the organic component is most vulnerable, then the inorganic, and is attacked by hydrolytic enzymes of microorganisms.

  Probably Staph. aureus is the microorganism, is involved in the process. (Pigmentation!)

- Proteolysis-chelation theory (Martin-Schatz 1955)

  Simultaneneous microbiological degradation of the organic components and the dissolution of the minerals by the process of chelation.

  Neutral or alkaline pH!

Endogenous theories:

- Proteolytic theory (Bodecker 1948)

  The mineralcontent of the pulpalymph is enough high to neutralize the lactic acid onto the tooth surface.

- Phosphate theory (Csernyei 1950)

  Caries started into the inner surface of the tooth, and this is a demineralisation. Causes: a changing in the pulp lymp for the effect of nerv system.

Exogenous theories:

- Sulfatase theory Pincus (1949)

  Proteolytic organisms first attack the protein elements. Sulfatases of Gram- bacilli hydrolyse mucoitin sulfatase of enamel and the chondroitin sulfatase of dentin and produce sulfuric acid.

- Complexing and phosphorilating theory (Eggers-Lura 1967)

  Uptake of phosphate by plaque bacteria occurs during glycolysis and synthesis of polyphos-phates.

Current concept of dental caries

Primary factors:
Initiate caries

Secondary factors:
Modify the progression

Bacterial plaque BIOFILM
- Oral hygiene, oral flora(quantitative and qualitative)
- saliva (pH, composition, buffering capacity, flow rate)
- Fluoride in plaque; Diet and nutrition;

Substrate
- Carbohydrates, (type and concentration)
- Chemical composition of food (fats, proteins)
- Physical characteristics of food (detergency, etc.)
- Oral clearance of food,
- Oral hygiene, frequency of eating

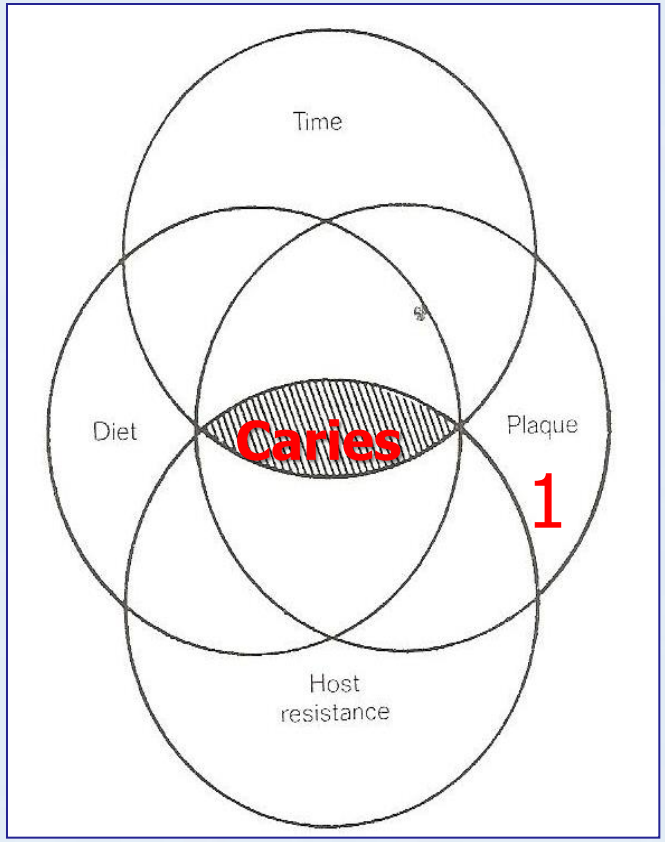
Tooth + TIME
- Fluoride concentration, carbonate and citrate level, age of tooth, gross and surface morphology (hypoplasia, fissures), Crystallinity of OH-apatite, Trace elements (Zn,Se,Sn,Fe,Mn,Mo) Nutrition: Vitamins and minerals, fats, protein, phosphates)
- Salivary composition and flow rate
- Surface composition of enamel

1.Bacterial plaque BIOFILM formation

Development:

- acquired pellicle: 0,1-1 µm (30min-1hour)
  originates from the salivary proteins
- Bacteria: Gram+ cocci, Gram -, aerob, anaerob
- Matrix (glukan)

Bacteria: Streptococcus mutans, mitis, sanguis,
Veillonella
Actinomyces



Difference in specific, (if causes caries)

non specific (every plaque causes caies) hypothesis!

2.Substrate

- **Praeresorptiv,** postresorptiv

- Carbohydrate

  highmolecular carbohydrate (starch)

  low-molecular carbohydrate

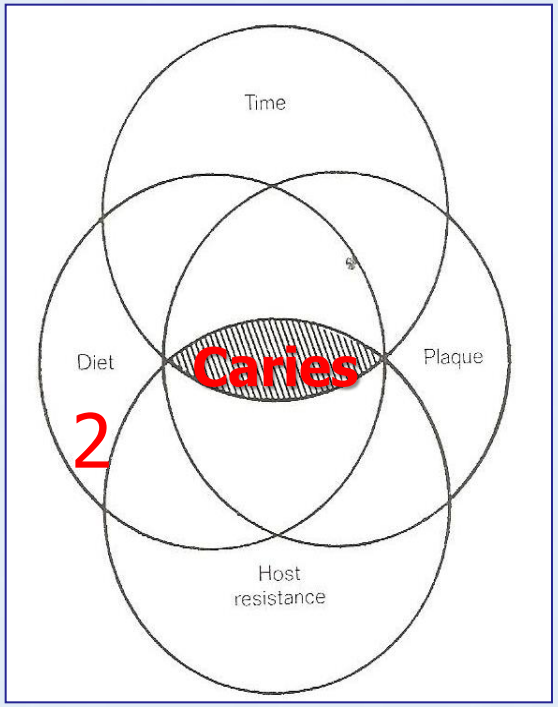
  saccharose (glucose + fructose)

  glucose, fructose

  organic acid:

  acetic-, lactic,- formic acid

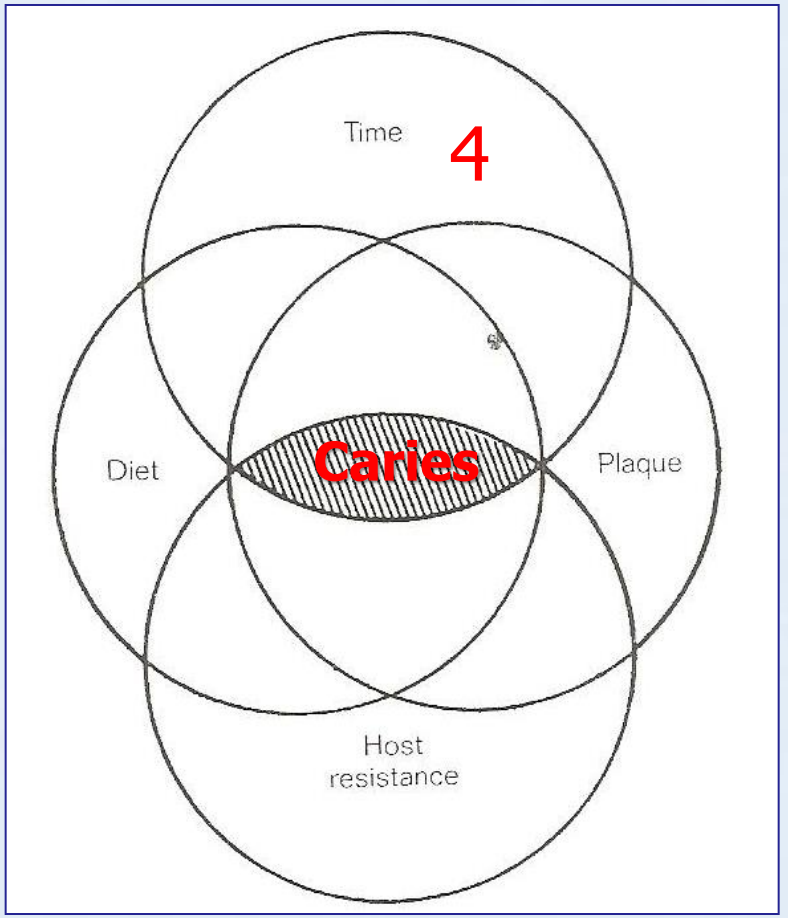
  pH-Value



The critical pH-value

for enamel is

5,2-5,7



3.Tooth

- Preeruptiv und

Posteruptive Maturation

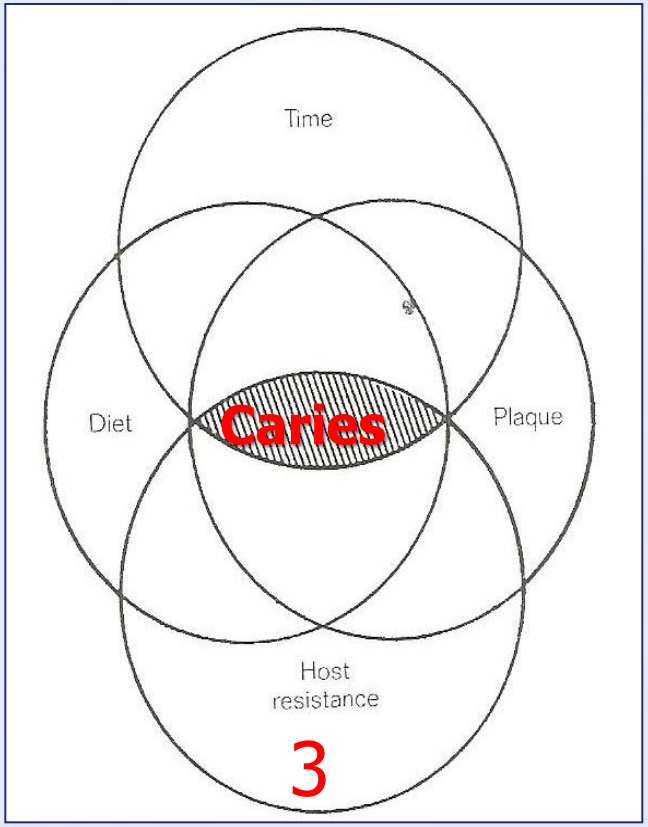
- Mineralisation (and structure) of the tooth

  before und after the eruption

  (preerupted and posterupted maturation!)

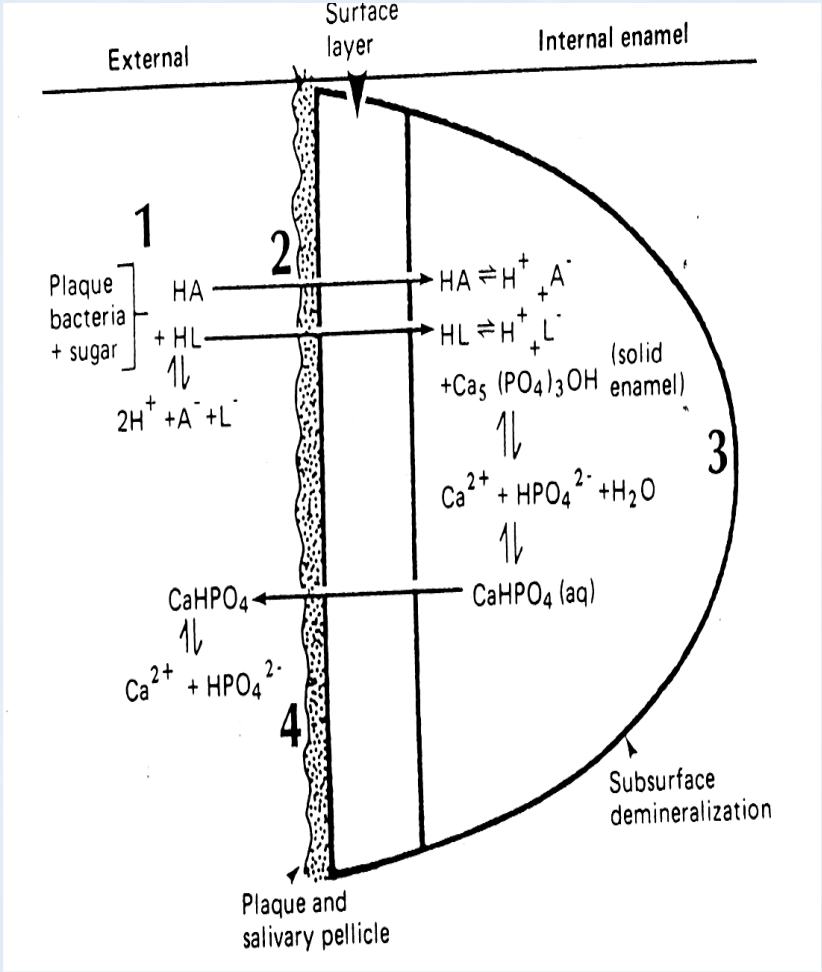
  Secondary factors are important!

- Caries predilection places



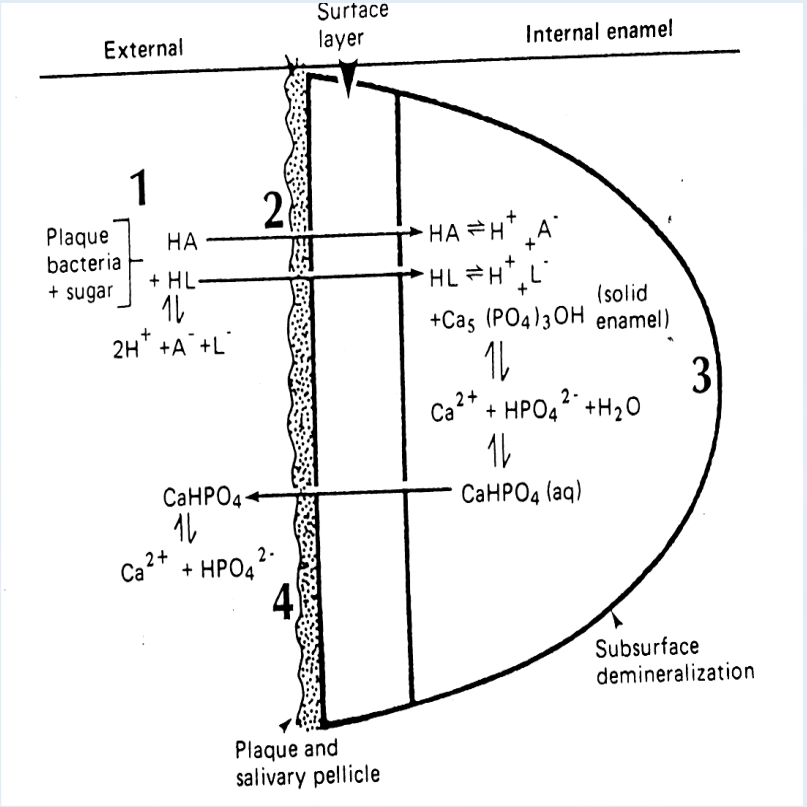
Enamel Caries Mechanism at a Chemical Level

- Primary factors and time are present.
- The reaction is controlled by the *diffusion.*
- The *H^+ ions* are the principal attacking species.
- The source of H^+ions is the *undissociated acid.*
  (acetic-, lactic,- formic acid)
- The weak, organic acid has *buffer function.*



Enamel Caries Mechanism at a Chemical Level

1. Organic acids will be produced.(Lactic, acetic, propionc)
2. The weak organic acids diffuse into the enamel. (concentrationsgradients!)
3. The weak acids can be present either in dissociated, or undis-sociated form. The H^+ attack the apatite cristals, and Ca^{++} Mg^{++} PO_4^{--} CO_3^{--} OH^- F^- will be free.
4. These Ions will diffuse to their concentrationsgradients to the external enviroment.

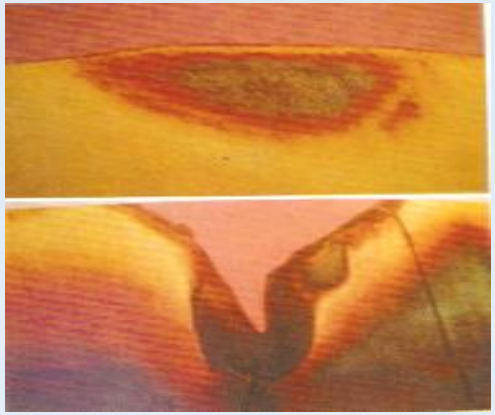


Demineralisation *or mineral loss remains, as long as sufficent acid is available.*

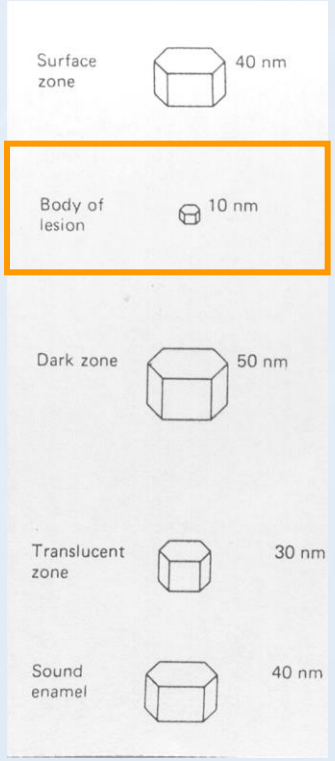
What is the consequence of this demineralisation?

- These reactions mostly occur in the demineralized subsurface layers particulary the body of the carious lesion, which may be as much as 70% demineralized

- The size of enamel crystal will be smaller. (10-30nm)



Layers of caries Incipient

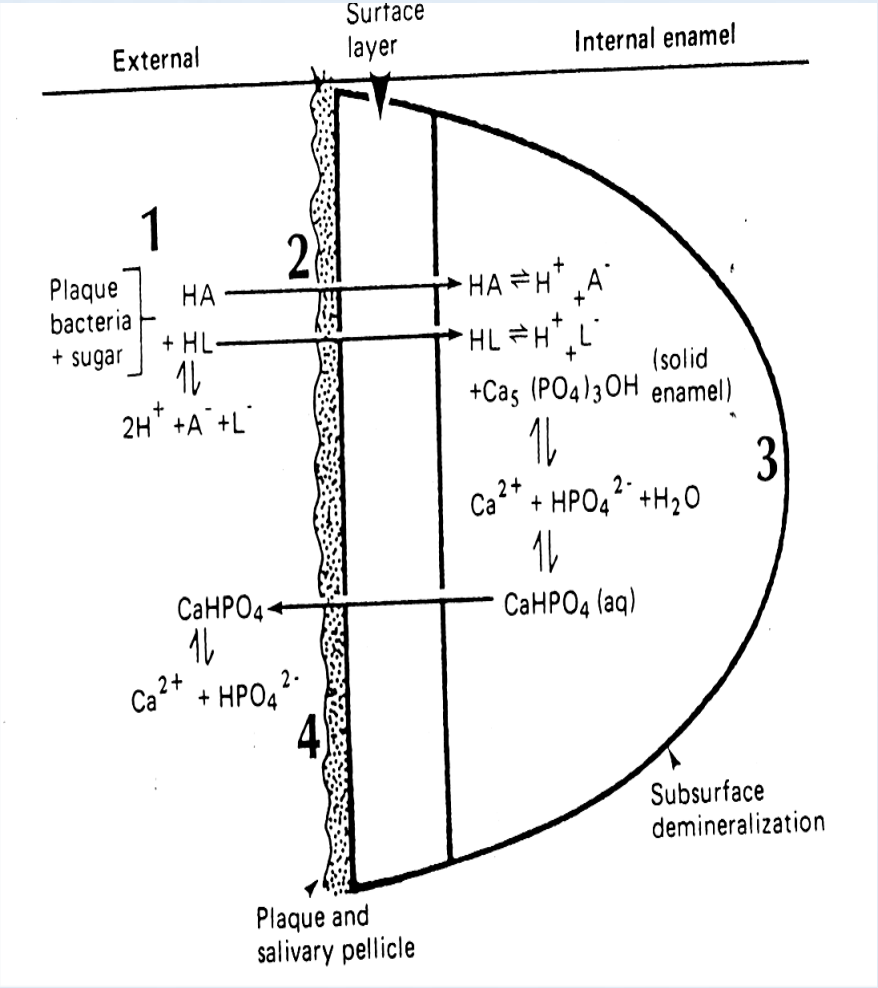


Enamel Caries Mechanism at a Chemical Level

4. When the acid product stops, and the concentration of Ca^{++} and PO_4^{-} increases, remineralisation may occur.

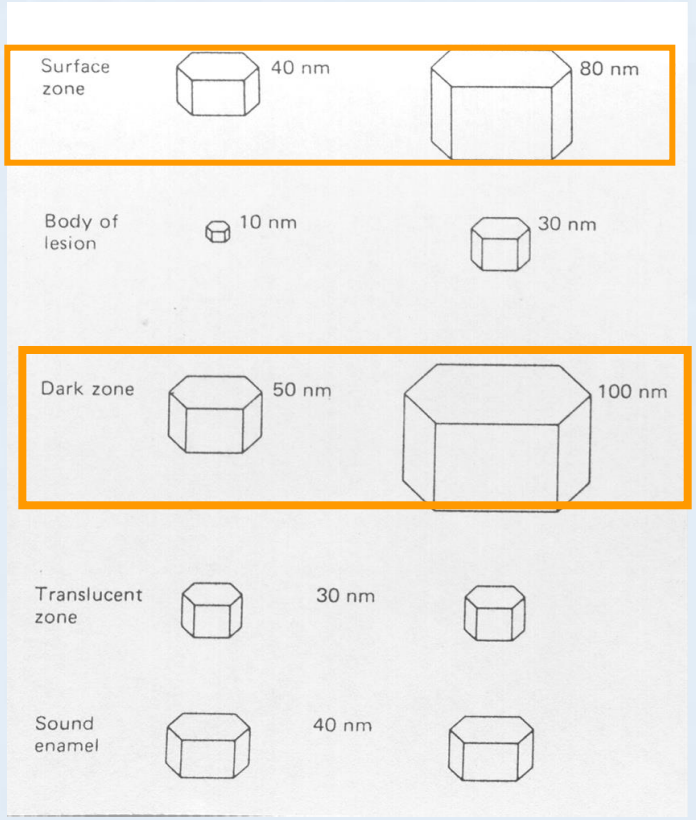
   Rekristallisation!

   (saturated solution)



What is the consequence of this remineralisation?

- As Ca^{++} and PO_4^{-} diffuse outwards, remineralisation becomes more and more likely as diffusion slows.
- The „surface zone „ (layer)and „dark zone" have been repaired,the crystals regrowth, the size of enamelprism: 80-100 nm



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Secondary Factors modify the progression