# Dental caries

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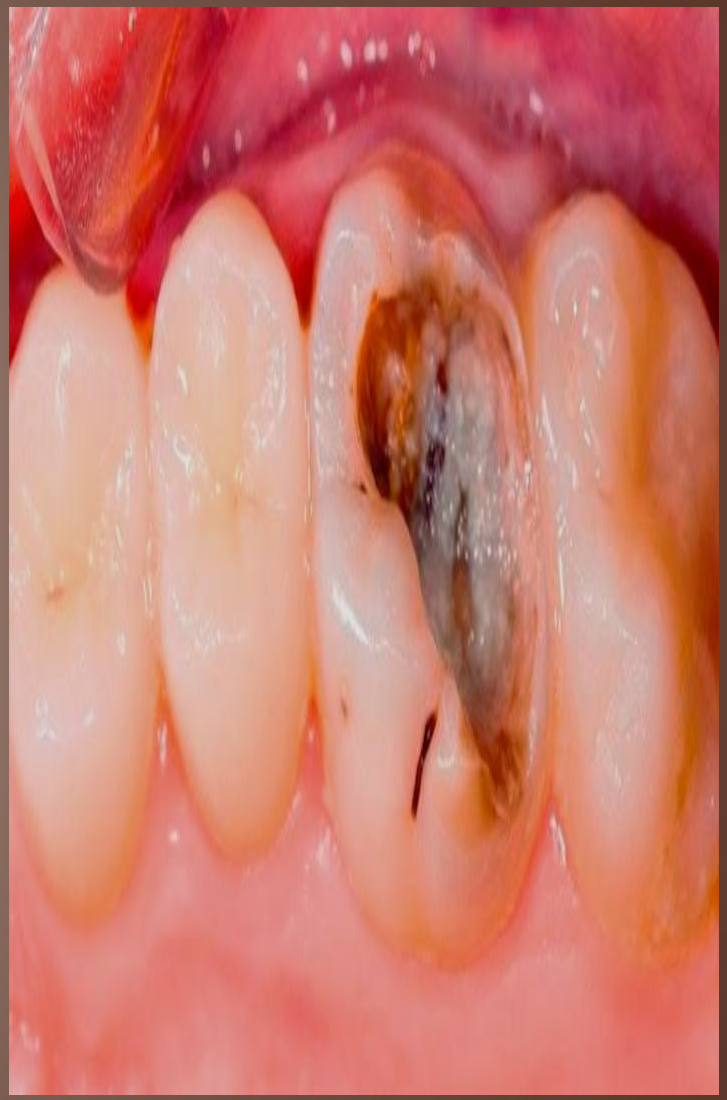
- It is a progressive bacterial disease of the calcified tissues of the teeth characterized by demineralization of the inorganic substance and destruction of the organic substance of the tooth to form a cavity.



# Causes of tooth decay:

- Bacterial causes
- Non-bacterial causes (attrition, abrasion, erosion and pathological resorption).

# Factors affecting caries prevalence:

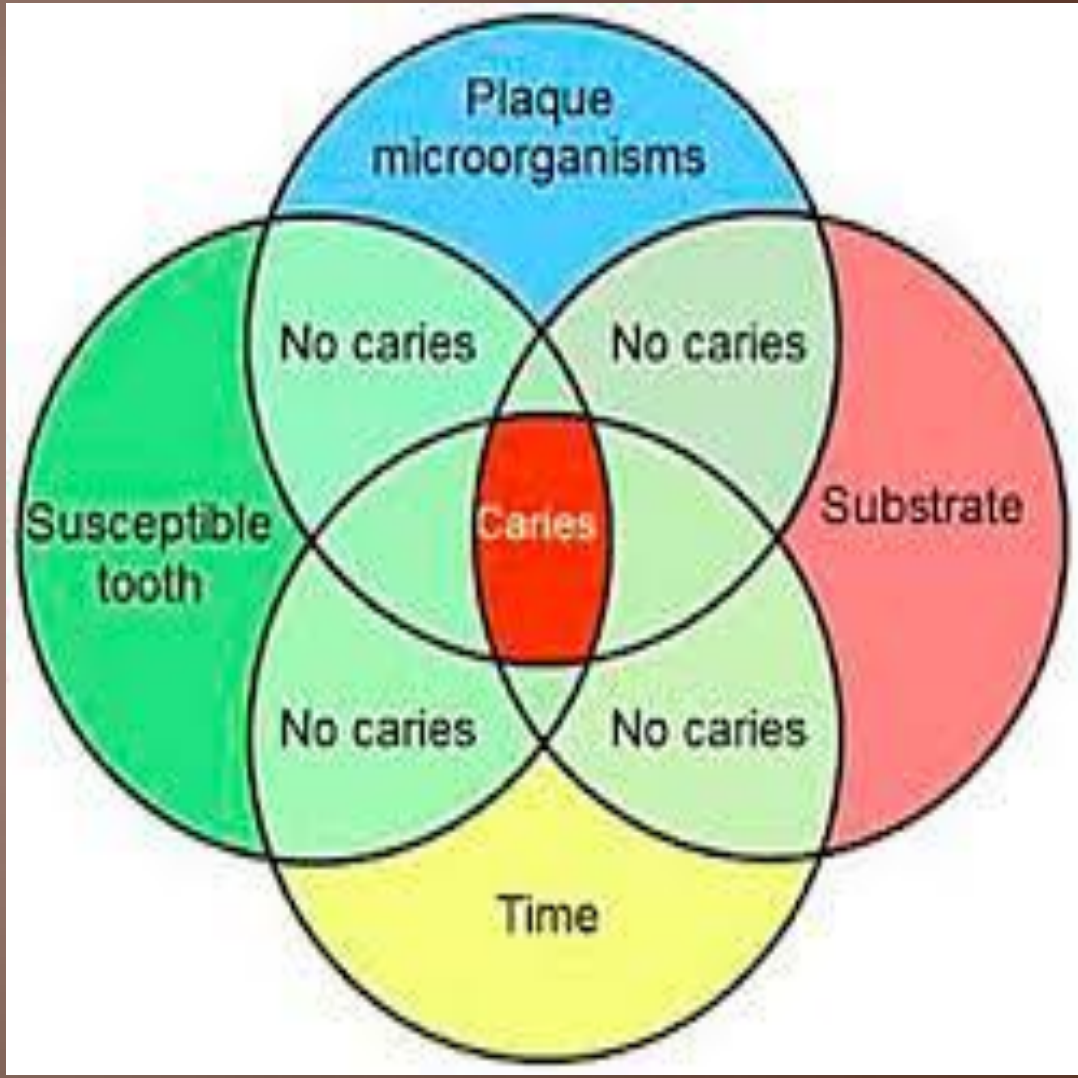
- **Race:** people living in the same geographical area but belong to different races have different caries incidence.
- **Age:** caries increase in old and children than middle aged.
- **Gender:** it is higher in female may be due to that their teeth erupt earlier compared to males.
- **Familial:** heredity tendency but influenced by local factors.

# Requirements of Dental caries

- It is multi-factorial infectious disease that requires the interaction of:
- **Host** (susceptible tooth surface and stagnation area)
- **Bacteria** (dental plaque) or bacterial plaque
- **Food** (dietary fermentable carbohydrates) or fermentable bacterial substrate
- **Time** to develop.

# Microbiology of dental caries:

- S. mutans and S. sobrinus (start the lesion)
- Lactobacilli (progressed the lesion)

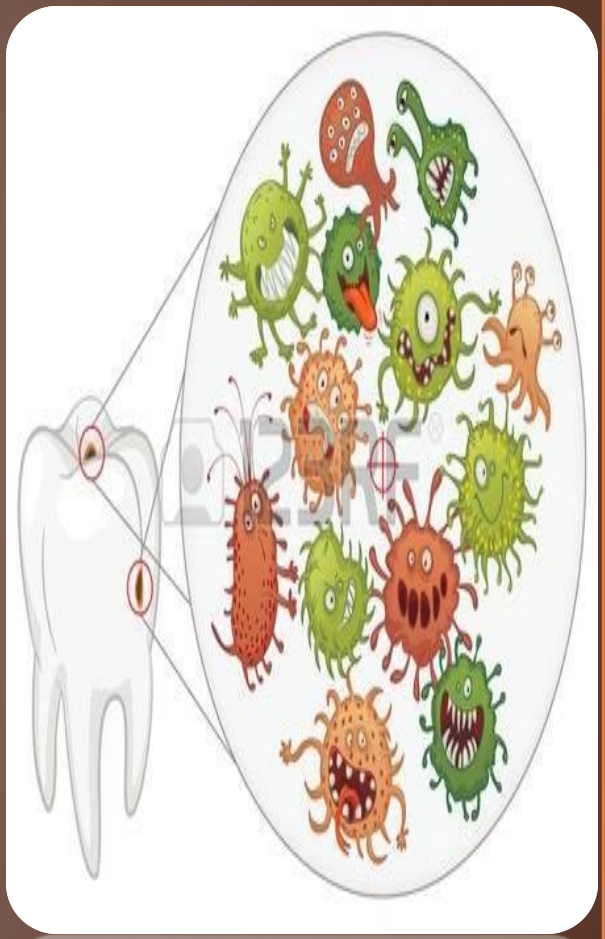


# Caries can be controlled by controlling the following:

- **Host:** administration of fluoride and fissure sealant
- **Micro-organism (bacterial flora):** active and passive immunity
- **Diet:** reduction in cariogenic sugar
- **Time:** control the frequency of sugar intake, ignore brushing 12-14 hours permit dental plaque development

# Theories for the aetiology of dental caries

- **Acidogenic theory (Miller`s chemoparasitic theory 1890):** most accepted theory
- proposed that dental caries develop in two phases:
- 1st phase bacterial attack inorganic
- 2nd phase disintegration of the organic matrix.



## Proteolytic theory (Bodecker 1878):

- it proposes that microorganism attacks the organic part of enamel leaving the generating acid responsible for further decalcification of inorganic part.
- **Proteolytic- chelation theory (Schatz 1955):** it proposes that caries occurred as a result of degradation of organic substance (proteolysis) and dissolution of tooth minerals by a process called chelation.

# Role of dental plaque:

- Dental plaque: soft, unmineralized bacterial deposit forms on teeth and dental prostheses.
- It is classified as:
- Supragingival plaque
- Subgingival plaque

# Acquired pellicle:

- layer covered tooth surface few seconds after cleaning consisting of saliva + microorganisms.
- There are synergistic or antagonistic bacterial interactions with variable bacterial composition at different sites of the teeth.

- Sugars are converted to lactic acid by bacteria and buffered saliva (The critical pH of 5.5).
- Fluoride ions inhibit bacterial metabolism by deposition of free mineral ions at the plaque. Systemic fluoride promotes the formation of stable hydroxyapatite crystals.

# Indirect factors influencing development and progression of dental caries include:

- **Factors intrinsic to the tooth:** enamel composition, enamel structure  hypoplasia and hypomineralization., tooth morphology, toothposition.
- **Factors extrinsic to the tooth:** Saliva (Flow rate, quantity, viscosity, PH, composition, presence of immunoglobulins and lysozyme), Use of fluoride, Immunity, Diet,  Physical factors ( Hereditary, Pregnancy and lactation).

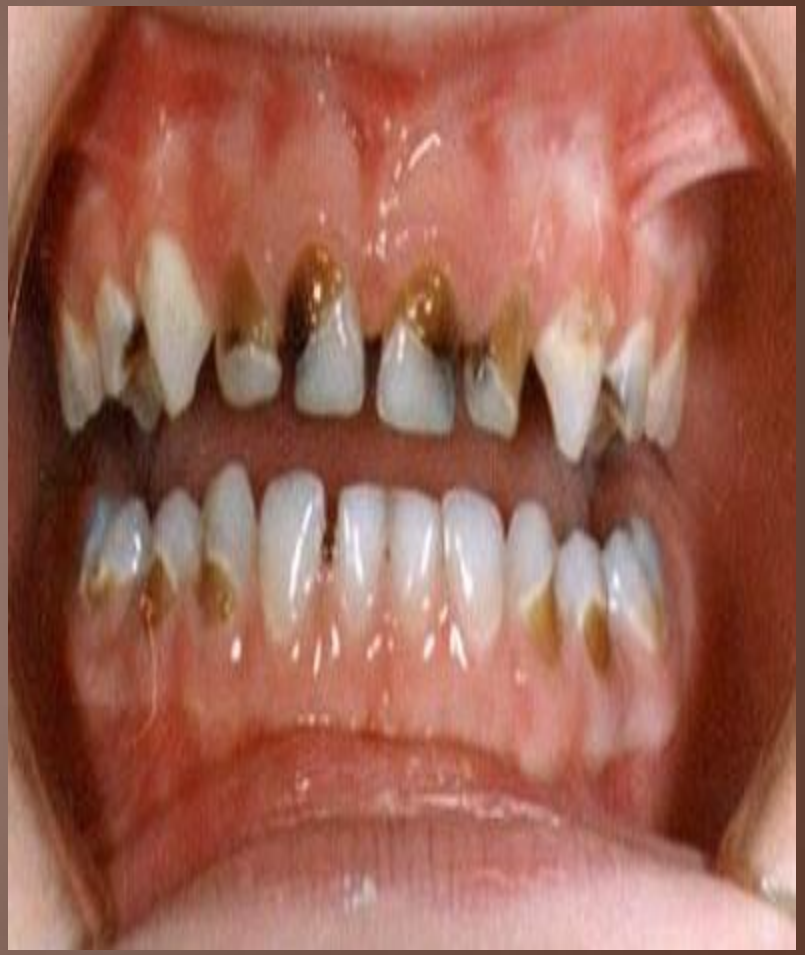
# Classification of dental caries

# According to the site of attack (location of the lesion) on the tooth

- Pit or fissure caries
- Smooth surface car.
- Cementam or root car.
- Recurrent caries

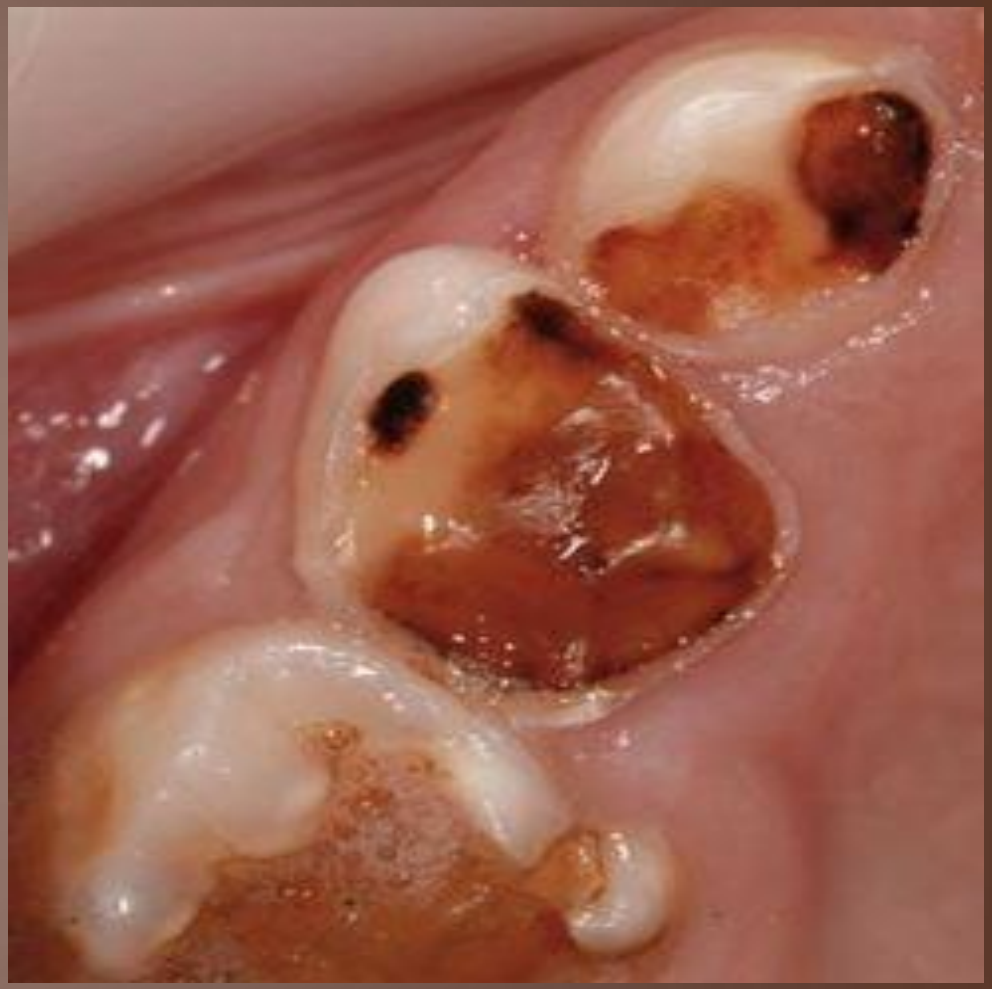
# According to the rate of attack

- **Acute caries:** rapidly progressive caries involving many erupted teeth.



- **Slowly progressive or chronic caries:** it is most common in adults and progresses slowly allow time for defence mechanisms of pulpodentinal complex to develop.

- **Arrested caries:** it is caries of enamel or dentine that becomes static and shows no tendency for further progression.



- **Rampant caries:** sudden rapid carious lesions of 10 teeth or more over a one year period observed in deciduous dentition of young children and permanent dentition of teenagers, diet and saliva are the most contributing factors in development.

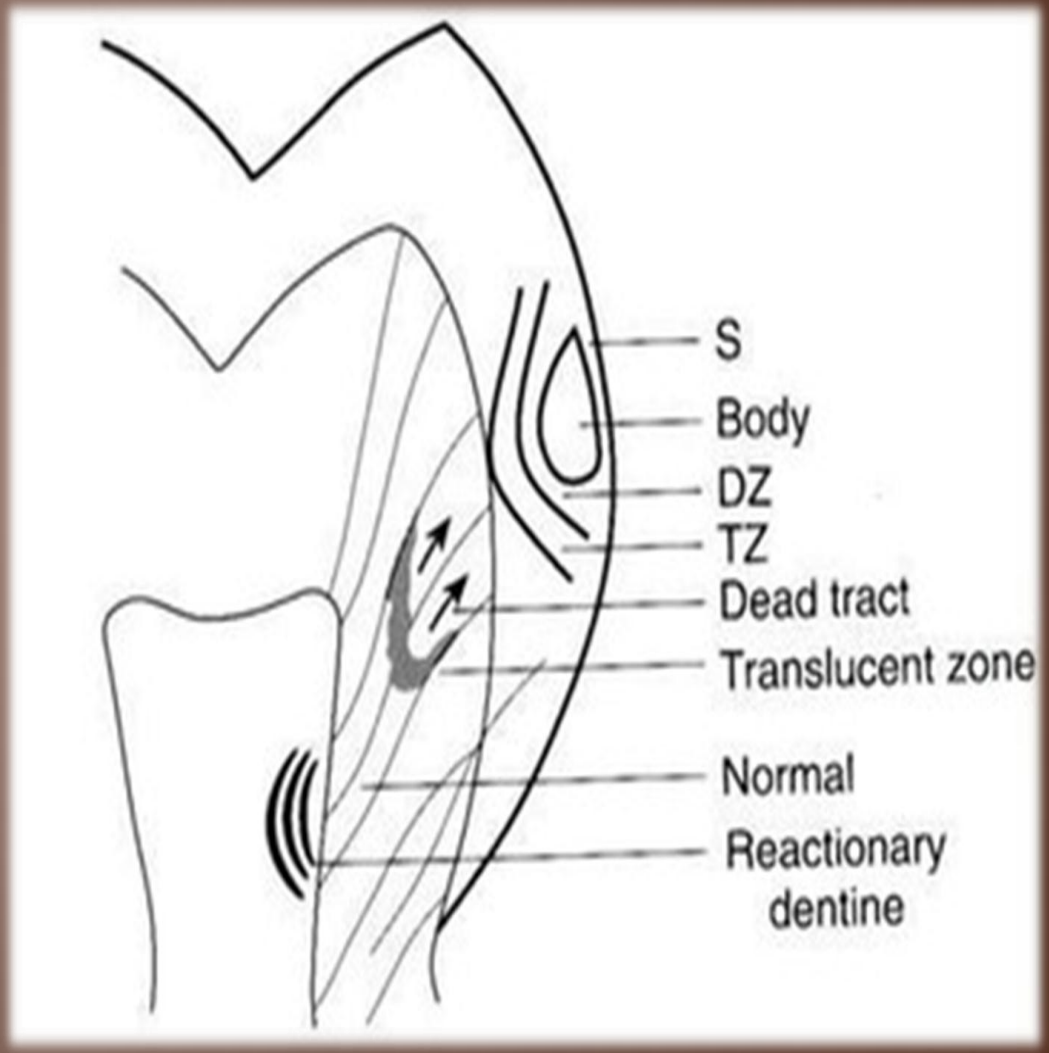
# Nursing bottle caries (Baby bottle syndrome and bottle mouth syndrome):

- Prolonged sucking of a sweetened drinks in a nursing bottle especially at night to about 2 years of age or beyond may be associated with rampant caries, involving particularly the smooth surfaces of the anterior maxillary teeth.

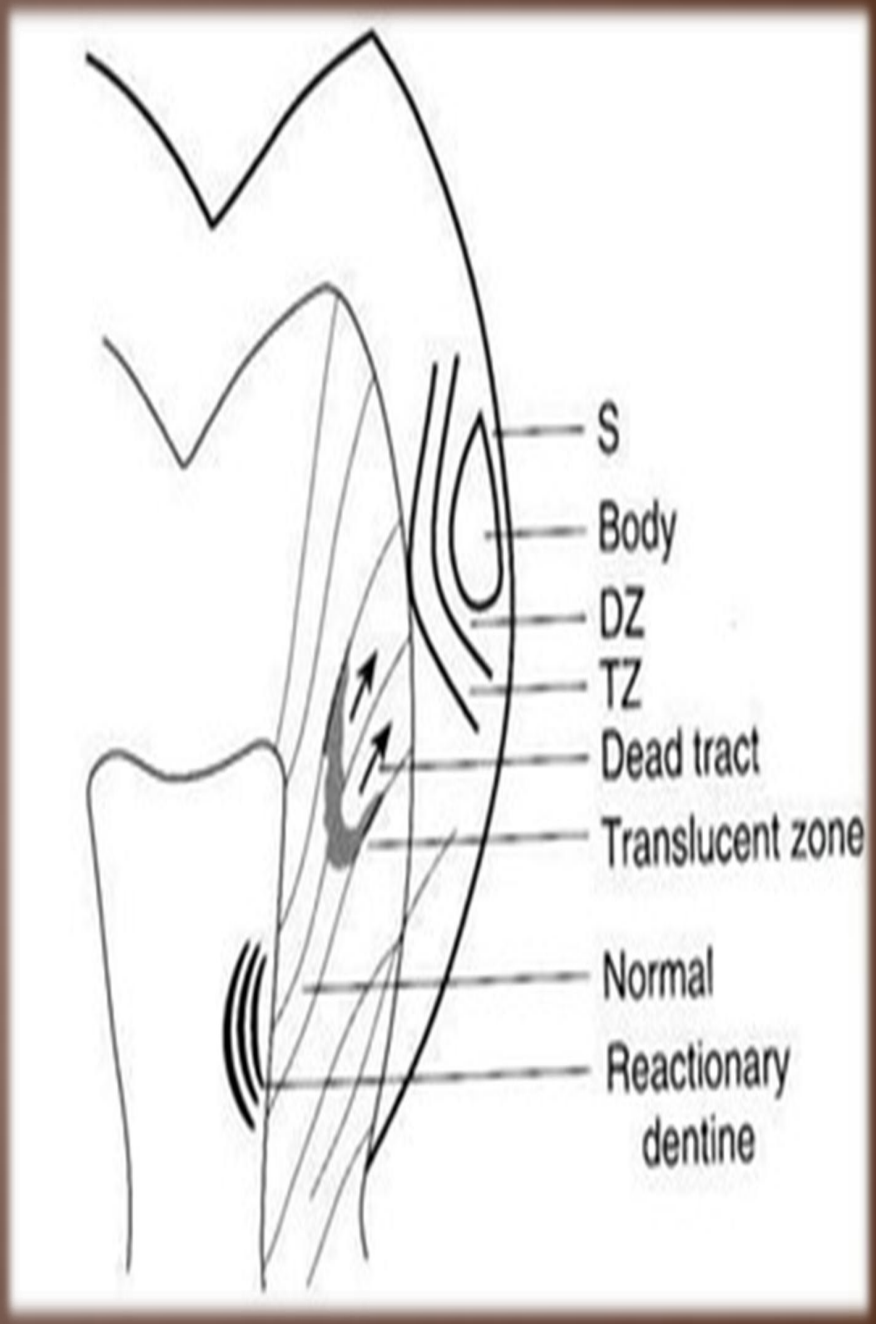
# Histopathology of dental caries

# Histopathology of enamel caries

- Cone-shaped lesion (base on the enamel and apex towards the amelo-dentinal junction.
- **Zone I (Translucent zone):** it is a subsurface layer where demineralization of magnesium and carbonate occurs.

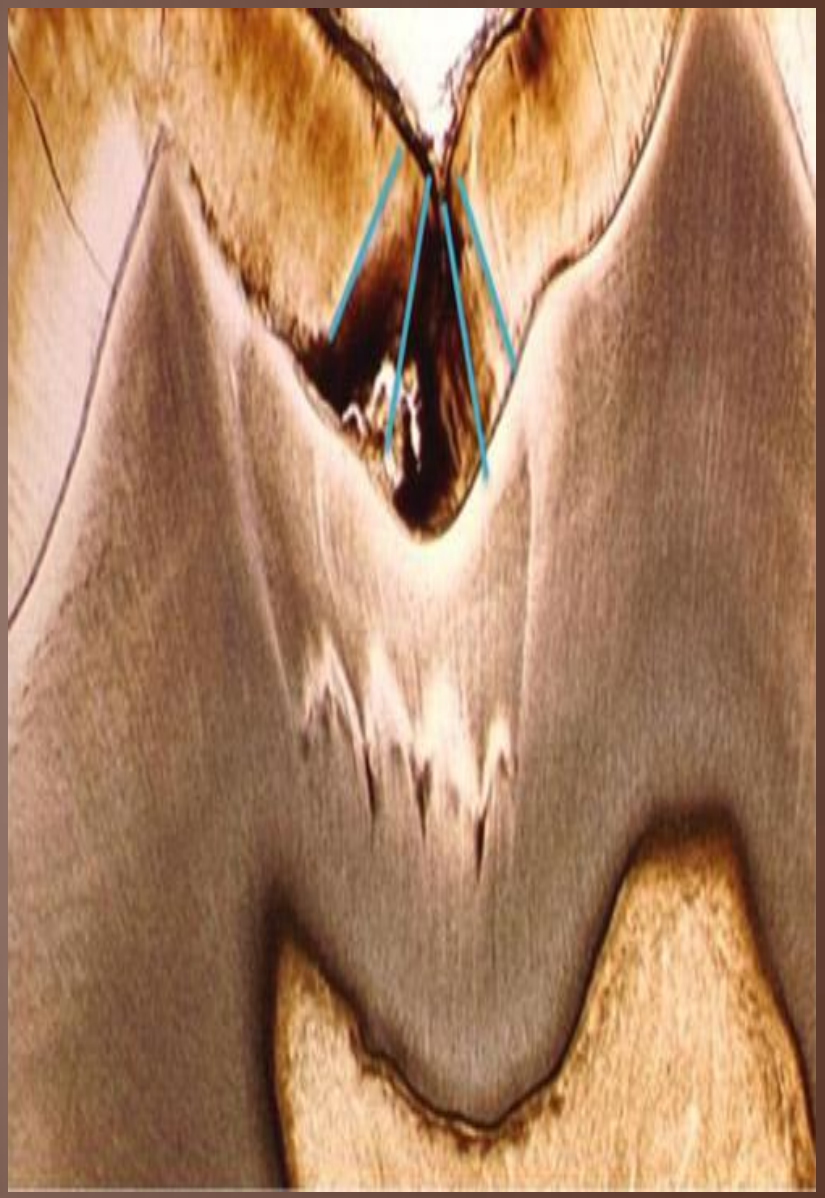


- **Zone II (Dark zone):** Some remineralization has occurred due to reprecipitation of mineral lost from the translucent zone. It is just superfacial to the translucent zone.

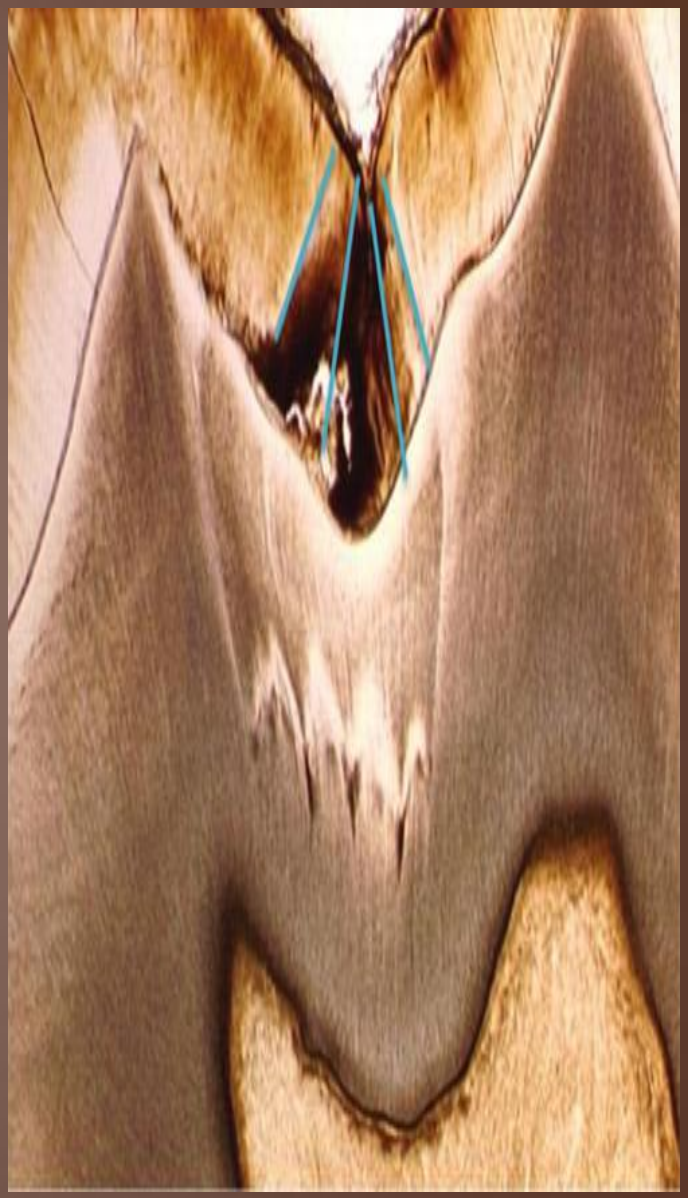


- **Zone III (Body of the lesion):** It is the largest portion of the lesion superfacial to the dark zone ,It is the area of greatest demineralization and having a higher fluoride level and lower magnesium level.

- **Zone VI (Surface zone):** The surface uneffected enamel have consists of high mineral and high fluoride level because it has reprecipitation of mineral derived both from the plaque and dissolved deeper areas.



- **Cavitation occurs more in pits and fissure than smooth surface caries because:**
- Enamel at the bottom of the pit and fissure very thin.
- Enamel rods are directed laterally (diverge) leading to the formation of cone shaped or triangle.
- Enamel at the surface become undermined and collapse under stress of mastication and fragmented.

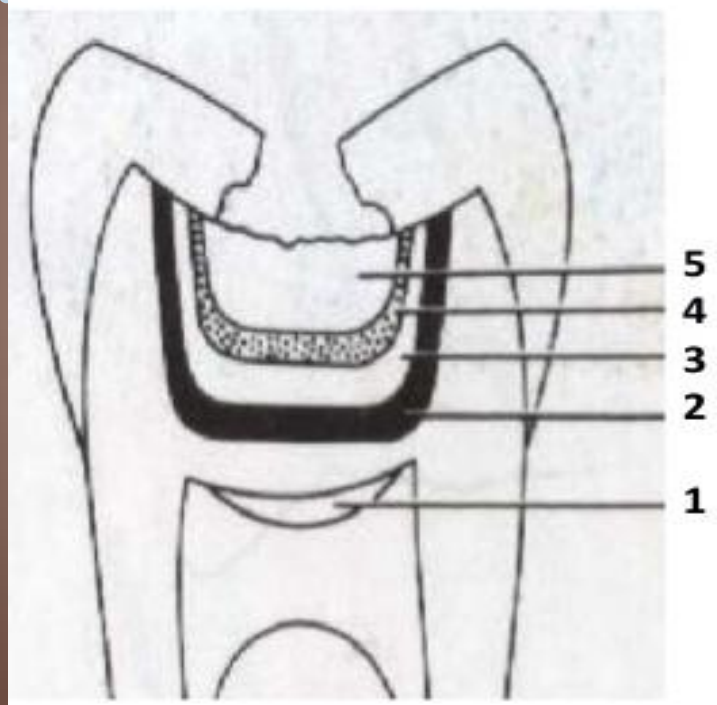


# Histopathology of dentine caries

- Dentine is a living tissue has higher organic content than enamel and a defenses mechanism to caries occur by:
- Development of dentinal sclerosis.
- Forming secondary dentine.
- Forming reparative or tertiary dentin.
- the lesion extened laterally in amelo-dentinal junction results in the involvement of great numbers of tubules and caries of dentine developed
- The early lesion is cone-shaped with the base at the amelo-dentinal junction.

# Zones of the dentine carious from the pulpal aspect:

- **Zone of fatty degeneration of protoplasmic process:** bacterial enzyme effect of the organic component.




Observing from the pulpal side at the advancing edge of carious lesion following different zones can be seen.

**ZONE 1**

Zone of fatty degeneration of Tomes' fibers

**ZONE 2**

Zone of dentinal sclerosis/Subtransparent dentin

**ZONE 3**

Zone of decalcification/Transparent dentin

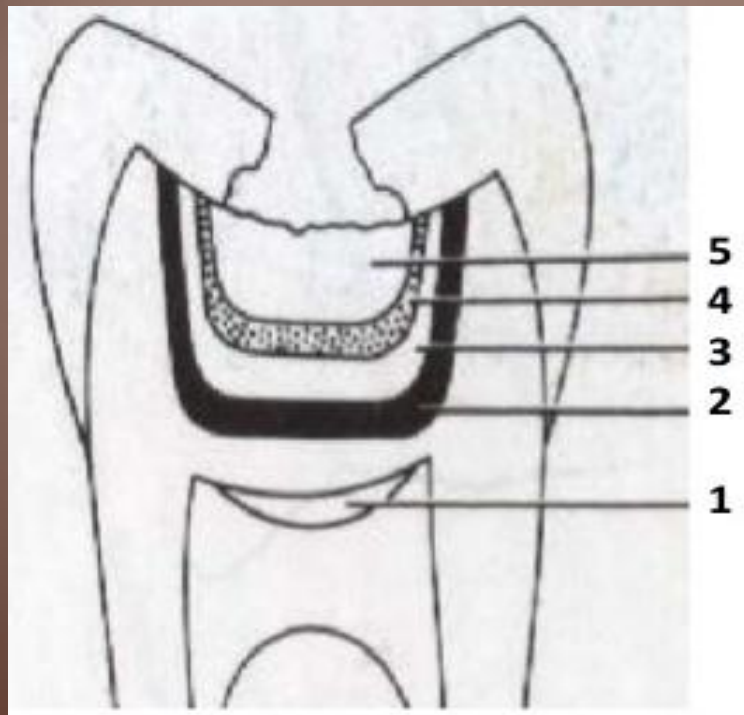
**ZONE 4**

Zone of bacterial invasion/Turbid dentin

**ZONE 5**

Zone of decomposed dentin/Infected dentine

- **Zone of sclerosis:** It is a vital reaction of odontoblasts to irritation (translucent zone) located beneath the carious lesion has higher mineral contents and Dead tracts may be seen.

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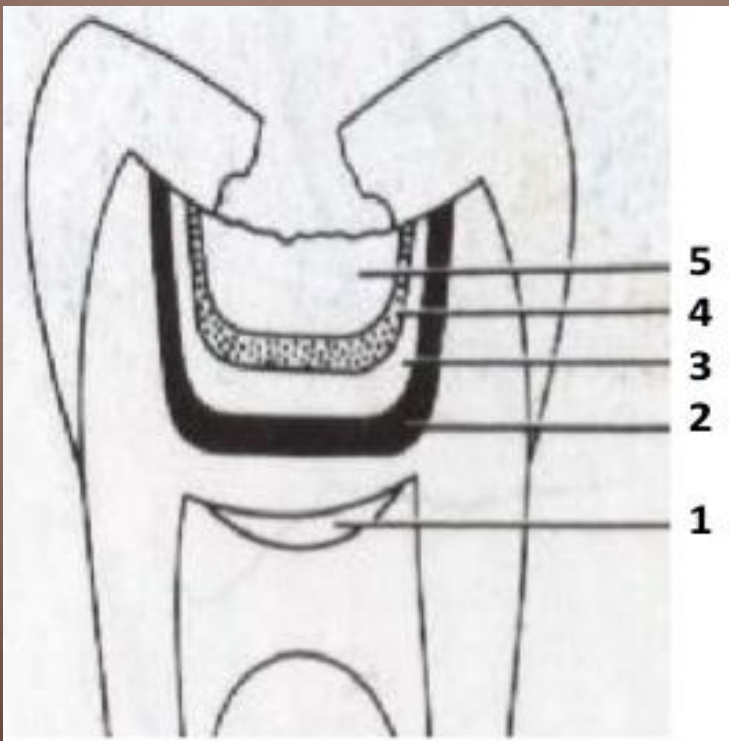
**Zone of decalcification (demineralization):** It is a softened dentine stained yellowish-brown as a result of the diffusion and interaction of bacterial products with proteins in dentine.

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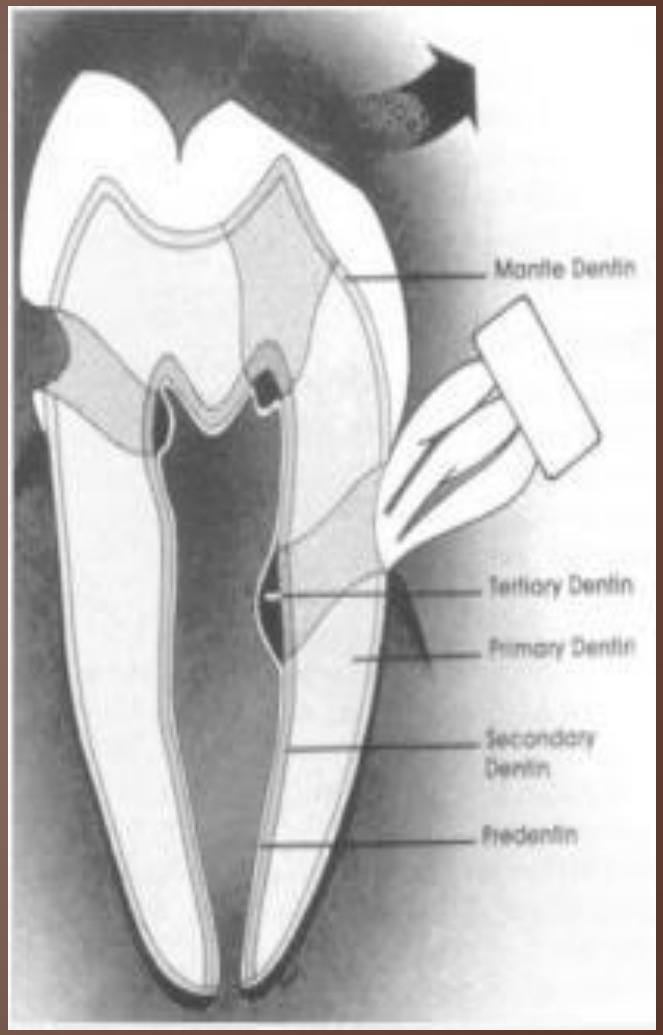
- **Zone of bacterial invasion:** mixed acidogenic and proteolytic bacteria occurred within the dentinal tubules resulting in areas of proteolysis-liquefaction foci.
- **Zone of decomposition or destruction:** area of liquefaction containing bacteria and necrotic tissue with little of the normal dentine architecture now remains.

# Reactionary (or tertiary)dentine

- Dentine layer formed as a non-specific response of irritated odontoblasts to caries, tooth wear, cavity and crown preparations.
- **Charactization of reactionary dentine:**
- Irregular
- Tortuous
- Hypermineralized comparing with primary dentine.



# Clinical aspects of dentine caries

- The soft stained or unstained dentine at the base of a cavity shoud be removed with **no** need to remove hard stained dentine that is **not** catch with probe
- or we used stepwise excavation of a deep carious lesion to remove infected dentine without pulpal exposure (sealing interval from 4 to 6 months) to allow time for the reactine dentin to develop.

# Immunological aspects of dental caries

- Humoral response against *S. mutans* is provided largely by secretory IgA antibodies in saliva which interfere the attachment of the organism to tooth surfaces, IgG and IgM antibodies can also gain access to the mouth via the crevicular fluid.

# Suggestive Reading

***Brad W Neville, Douglas D Damm, Carl M. Allen, Jerry E Bonguot. Oral And Maxillofacial Pathology, 4th Edition, Elsevier, 2015***


Thank
You!