

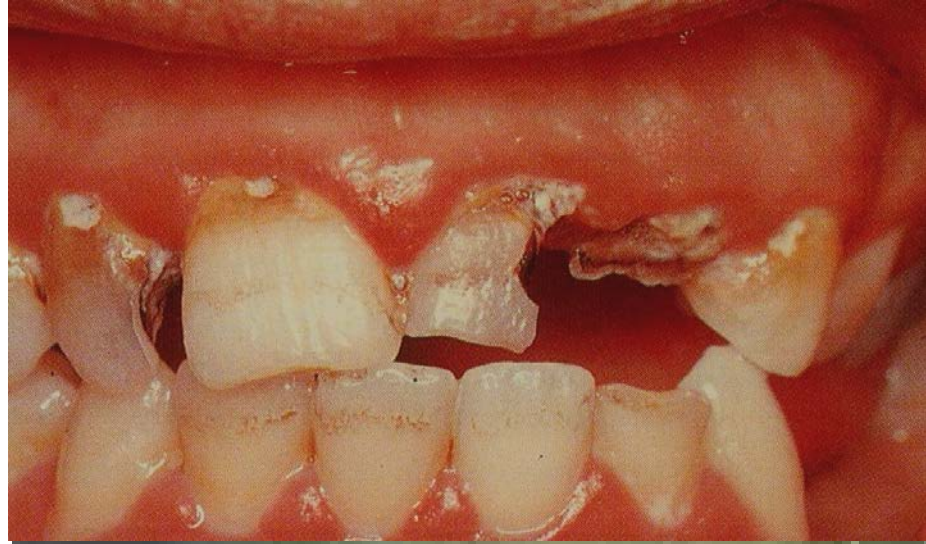
DENTAL CARIES

DENTAL CARIES

DEFINITION

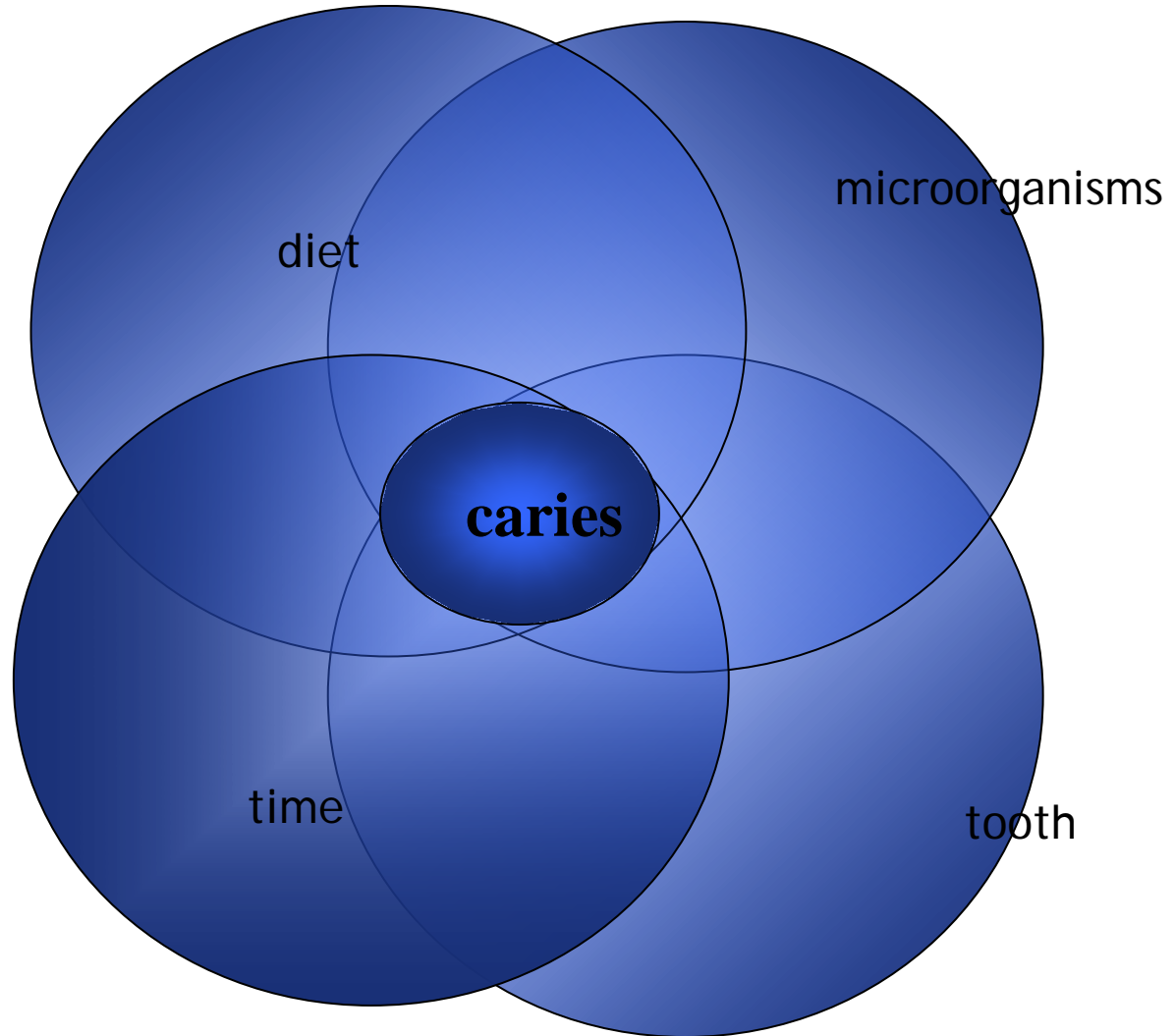
It is a disease of microbial origin in which the dietary carbohydrates are fermented by the bacteria forming an acid which causes the demineralization of the inorganic part and ◉ disintegration of the organic part of the tooth.

ETIOLOGY



- Dental caries is a multifactorial disease.
- Involves demineralization of the enamel and dentin.
- Bacteria in dental plaque fermentable carbohydrates such as sugar (sucrose) producing organic acid.
- Continuous acid production causing plaque pH to fall below 5.
- Repeated fall in pH with time, causing demineralization of susceptible site on the tooth surface, initiating a carious process.

Etiology



Diet+ Bacterial flora → Acid + Tooth → D.C

THEORIES OF DENTAL CARRIES

The Acidogenic theory

The Proteolytic theory

Proteolysis chelation theory

ACIDOGENIC THEORY

- W.D Miller was the first well known scientist and investigator of dental caries and published his results in 1890.
- It is the more accepted & supported theory
- In germ-free oral hygiene laboratory animals= administration of sugar → No dental caries
(Dental caries produced by chemical action of m.o)
- According to him the dental decay is a **chemoparasitic** process.
- It is a **two** stage process :There is decalcification of the enamel which also results in the destruction of the dentin. In the second stage there is dissolution of the softened residue of the enamel and dentin.
- Inorganic decalcification → Acid → organic dissolution
(m.o)

The Proteolytic theory : (Bodeker 1878)

Another theory was proposed which claims that the organic portion of the tooth is attacked first with lytic enzymes. This leaves the inorganic portion without a matrix support causing it to be washed away creating cavities.

M.O → E lamella & interprismatic substance

PROTEOLYSIS - CHELATION THEORY

- Schatz *et al* in 1955 proposed that caries occurred as a result of simultaneous degradation of organic substances (Proteolysis) and dissolution of tooth minerals by a process called **Chelation**

Initial attack on organic component of E → proteolysis

breakdown product → chelating with mineralized

component of E → decalcify E even at neutral or alkaline ph

ESSENTIAL PROPERTIES OF CARIOGENIC BACTERIA

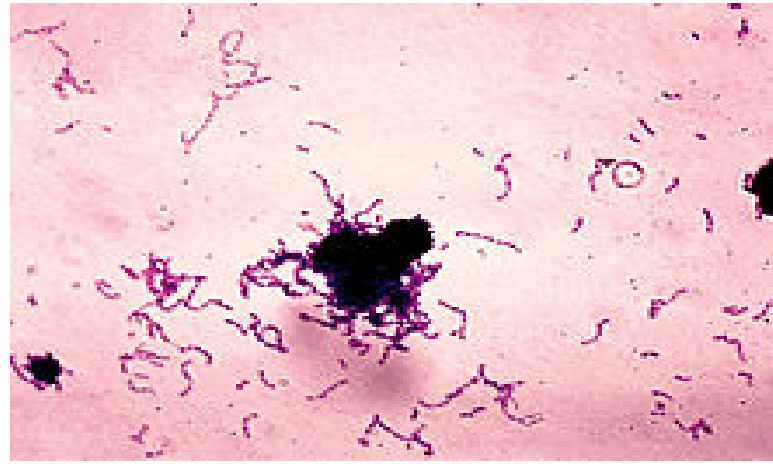
- ◉ • Acidogenic
- ◉ • Able to produce a pH low enough (usually $\text{pH} < 5$) to decalcify tooth substance
- ◉ • Able to survive and continue to produce acid at low levels pH
- ◉ • Possess attachment mechanisms for firm adhesion to smooth tooth surfaces
- ◉ • Able to produce adhesive, insoluble plaque polysaccharides .

MICROBIOLOGY of D.C

Flora Consists of micro-organisms which are brought in the contact of the tooth via plaque.

Micro-organisms mainly associated are :-

- (1) Streptococci (*S. mutans*, *S. salivarius*,
S. mitis, *S. sanguis*, *S. sobrinus*)
- (2) Lactobacilli (*L. acidophilus*)-
secondary invaders
- (3) Actinomyces (*Viscosus*)



Role of carbohydrates

- Caries prevalence increased when population exposed to sucrose-rich diets.
- Extrinsic sugar are more damaging than intrinsic sugars.**
- Sucrose is the most cariogenic sugar because:
 - 1-It is readily fermented by plaque bacteria.
 - 2-It is easily converted to extracellular glucans by bacterial glucosyl transferase.
- Frequency of sugar intake is of more importance than total amount consumed.**

AETIOLOGICAL VARIABLES

(INDIRECT FACTORS, CONTRIBUTING FACTORS)

Factors intrinsic to the tooth:

- 1- Enamel composition:- Inverse relationship between enamel solubility & enamel fluoride concentration.
- 2- Enamel structure:- Developmental enamel hypoplasia & hypomineralization may affect the rate of progression but not the initiation of caries.
- 3- Tooth morphology:- Deep narrow pits & fissures favour the retention of plaque & food.
- 4- Tooth position:- Malaligned teeth may predispose to the retention of plaque & food.

AETIOLOGICAL VARIABLES

Factors extrinsic to the tooth:

- 1-Saliva:-Composition(inorganic constituent), Quantity (flow rate), Viscosity, Buffering capacity (PH) , The antimicrobial agents present in saliva (IgA prevent proliferation of bact).
- 2-Diet:-Frequency of intake , State of diet, Vitamin content of diet (Vit D deficiency causing enamel hypoplasia--- Vit K utilized as anticariogenic agent as having enzyme inhibiting action in carbohydrate degradation.
- 3- Use of fluoride:- F⁻ readily enters bacterial cells & can inhibit enzymes involved in the metabolism of sugar.
- 4- Immunity:-IgA in saliva & IgG in gingival crevicular fluid.

Pathology of dental caries

Dental caries classified clinically by:

- ◎ **Site of attack**
 1. **Pit and fissure**
 2. **Smooth surface**
 3. **Cemental or root caries**

DENTAL CARIES CLASSIFIED CLINICALLY BY

◉ Rate of attack

1- Acute (rampant)

2- Chronic

3- Arrested

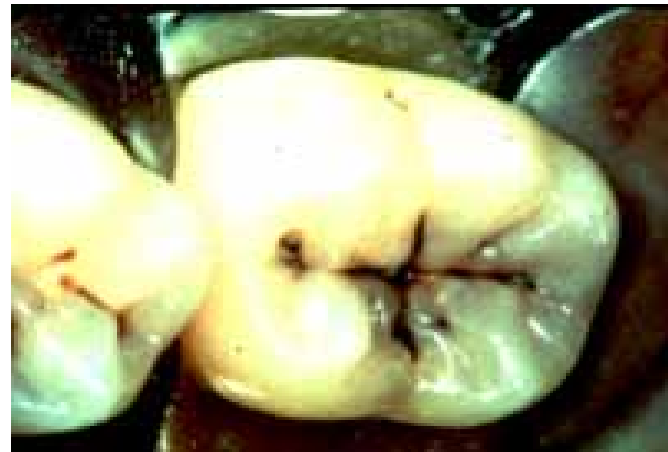
- According to whether:

1- The lesion is a new
(primary , virgin caries)

2-Under previous restoration
(secondary , recurrent caries)

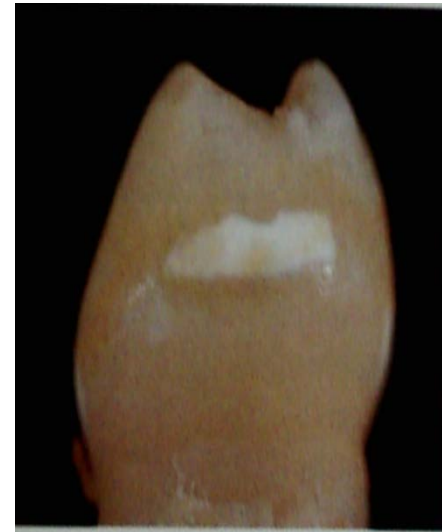
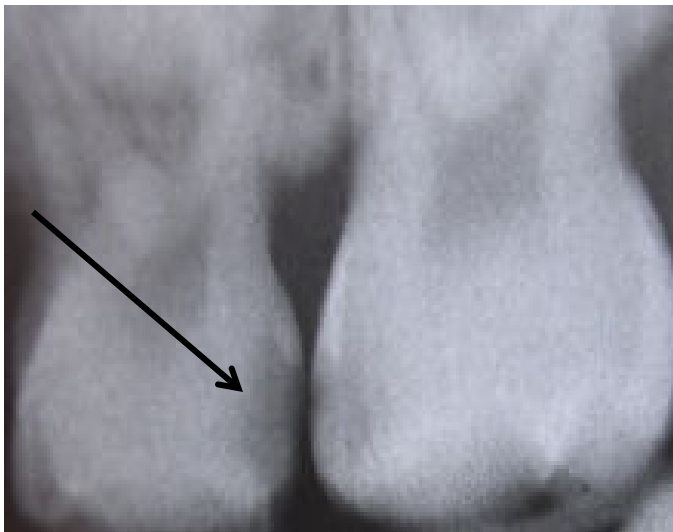
◎ 1- Pit and fissure caries

- ◎ Found mainly on the occlusal surfaces of posterior teeth, buccal & lingual surfaces of molars, and in lingual (palatal) pits of maxillary incisors.
- ◎ Early lesion appear as a brown or black discoloration of a fissure, probe stick to it
- ◎ Enamel appear opaque, bluish- white when undermined by caries.



◎ 2- SMOOTH SURFACE CARIES :

- ◎ **SMOOTH SURFACE CARIES** develops on proximal surfaces of all teeth or on gingival 1/3rd of buccal and lingual surfaces.
- ◎ Occur below contact point . The initial lesion appear as a well-demarcated chalky-white opacity of enamel without continuity loss, & it is not detected by probe or by routine radiograph.
- ◎ In progressive lesion:-Enamel appear bluish-white , rough surface , detected by probe, on radiograph appear as R.L area.



Cervical caries:- appear as a proximal one but with a wide open cavity.



3-CEMENTAL CARIES

Occur due to root exposed as a result of periodontal diseases. (Gum recession)

Appear as a shallow, saucer-shaped , with ill-defined boundaries.



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- For root caries to begin, formation of dental **plaque** and bacterial invasion are essential.
- However, the microflora may be different for root caries compared to coronal caries. Most commonly the invading bacteria are **filamentous** rather than cocci.
- Bacteria appear to invade either along **Sharpey's fibers** or between the fiber bundles just like invasion of **dentinal tubules**.
- Once, dentin is involved, progression is similar to coronal caries but for a **slower rate** of progression

RAMPANT (ACUTE) CARIES

- Extensive rapidly progressing caries affecting many teeth (primary & permanent), --Larger D.T, No sclerosis
 - Light yellow color Vs brown\ black in chronic one
 - Painful
- 10** or more carious lesion over 1 year period may occur
- e.g (bottle feeding carie)**
- An early pulpal involvement.
- (no time for pulpo-dentinal complex formation)--No sclerosis



SLOWLY PROGRESSING (CHRONIC) CARIES

- ◉ Progress slowly.
- ◉ Mild pain.
- ◉ Later pulpal involvement.
- ◉ Common in adult.
- ◉ Pulpo-dentinal complex develop.

ARRESTED CARIES



- It is that caries which becomes static or stationary and does not progress any further.
- Can occur in both deciduous and permanent dentition.
- Occurs almost exclusively on occlusal surface.

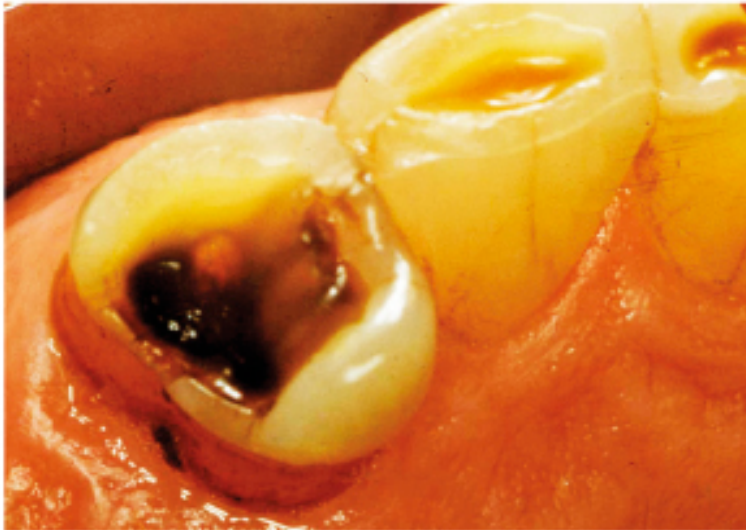


Figure 17. Clinical illustration of arrested caries. The dentin is hard, darkly discolored, dry looking and plaque free.

4-RECURRENT CARIES

Occurs around the margin or at the base of an existing restoration.

Usually due to inadequate extension of restoration resulting in food impaction

