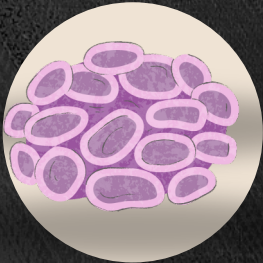
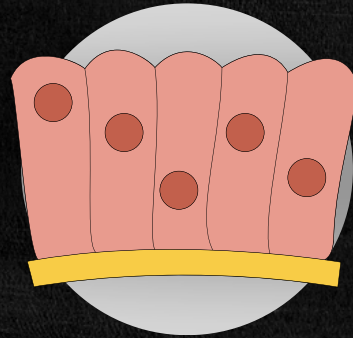


# Oral Pathology



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## Definition of Dental Caries & Role of Carbohydrates :

**Definition :** Dental caries is an irreversible microbial disease of calcified dental tissues characterized by:

- Demineralization of the inorganic portion.
- Destruction of the organic substance.
- May eventually lead to cavitation.

It is a chronic, progressive, destructive disease of hard dental tissues and is considered a post-eruptive pathological process of external origin.

### **Role of Carbohydrates :**

Carbohydrates are the main dietary component utilized by cariogenic bacteria to produce acids.

#### **Types**

##### **Monosaccharides:**

- Glucose
- Galactose
- Fructose
- Ribo

##### **Disaccharides:**

- Sucrose
- Lactose
- Maltose

##### **Polysaccharides:**

- Starch
- Cellulose
- Glycogen

### **Why is Sucrose Highly Cariogenic?**

Because it:

- Has low molecular weight → rapidly diffuses into plaque.
- Is easily fermented by cariogenic bacteria → acids such as lactic and pyruvic acid.
- Helps bacteria form extracellular polysaccharides, especially dextran.
- Is cheap and consumed frequently.

### **Factors Increasing Cariogenicity**

- ↑ Total amount of carbohydrate.
- Sticky/retentive consistency.
- ↑ Frequency of intake.
- High refinement.

**Important :** Frequency is especially important because every carbohydrate exposure can prolong the period of bacterial acid





## Role of Bacteria & Dental Plaque

### Historical Evidence

- Miller (1890): proposed that bacteria ferment carbohydrates → acids → caries.
- Orland et al. (1905): germ-free rats did not develop caries despite a cariogenic diet; caries developed after bacteria were introduced.

### Characteristics of Cariogenic Bacteria

They should be:

- Acidogenic: produce acids from fermentable carbohydrates.
- Aciduric: survive in acidic environments.
- Able to produce extracellular polysaccharides → adhesion and plaque formation.
- Able to store intracellular carbohydrates.

#### 1- Streptococcus mutans

- Major organism in initiation of dental caries.
- Acidogenic + aciduric.
- Produces dextran, levan and amylopectin.
- Can cause smooth-surface and pit/fissure caries.
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#### 2- Lactobacilli

- Important in progression, especially dentinal caries.
- Acidogenic and aciduric.
- Cannot synthesize extracellular polysaccharides effectively → cannot initiate smooth-surface caries alone.
- Their number increases in an already acidic carious environment.
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#### 3- Actinomyces

- Particularly associated with root-surface caries.
- Proteolytic gram-positive organisms.





### ▪ Symbiosis:

Different organisms can support each other metabolically.

For example:

Streptococci → polysaccharides → disaccharides → yeast → monosaccharides → Lactobacilli → acids

### Dental Plaque

Dental plaque is a tenaciously adherent bacterial structure on tooth surfaces.

### Composition

60–70% microorganisms

- Mainly streptococci
- Lactobacilli
- Actinomyces

30–40% amorphous matrix

- Glycoproteins
- Epithelial cells
- Amylase
- Immunoglobulins

### Also contains

- Extracellular polysaccharides: dextran + levan
- Intracellular polysaccharide: amylopectin
- Lipids
- Ca, phosphate, potassium
- Fluoride

## Plaque formation

### 1. Acquired Enamel Pellicle

Bacteria-free salivary glycoprotein film.

About 0.3–1  $\mu\text{m}$ .

Forms rapidly after cleaning the tooth.

### 2. Initial Colonization

Within minutes to hours:

- sanguis
- oralis
- mitis

are important pioneer organisms.

Adhesins attach bacteria to pellicle





### 3. Growth Phase

Approximately 8–48 hours.

Bacteria multiply and form a monolayer.

Growth becomes perpendicular to the tooth surface.

### 4. Intermediate Community

Secondary colonizers appear.

*S. mutans* increases.

Acid production and extracellular polysaccharide formation increase.

### 5. Mature Plaque

Around 2 weeks or more.

Characteristic “corn-cob” structures may appear.

*Actinomyces* becomes an important component.

This change in microbial composition is called microbial succession.

## Stephan Curve / Plaque pH & Contributing Factors

### Stephan Curve / Plaque pH

After carbohydrate intake:

Sugar → rapid bacterial metabolism → acid production → rapid fall in plaque pH

Salivary buffering + removal/metabolism of acids → gradual pH recovery

The critical concept is that plaque can maintain an acidic environment longer than free saliva.



## Contributing Factors

### A. Tooth

Enamel resistance is related to:

- High mineralization.      Fluoride incorporation.
- Lower water/organic content.      Other mineral elements.

### Tooth Morphology

Higher susceptibility with:

- Deep pits.      Deep/narrow fissures.
- Buccal/lingual pits.
- Hypoplasia.
- Hypocalcification.
- Hypocalcification.

### Tooth Position

Malalignment and malocclusion → food/plaque retention → ↑ caries.

### B. Saliva

Important protective effects:

#### Remineralization

Mainly through:

- Calcium    Phosphate      Buffering    Ammonia and urea help neutralize acids

#### Antibacterial Action

Includes:

- IgA      IgG
- I gM      Lysozyme
- Lactoferrin
- Lactoperoxidase

Normal salivary pH is around 7.2.

↓ Salivary pH → favors demineralization.





### C. Diet

More cariogenic:

- Frequent sugars.      Refined carbohydrates.      Sticky foods.

Less cariogenic:

- Unrefined foods.      Fibrous foods.      Foods rich in fat/cellulose.

Important Nutritional Factors:

- Calcium      Phosphate      Vitamin D      Vitamin B6
- Vitamin K      Fluoride

Fluoride in drinking water is particularly important for caries resistance.

### Classification of Caries: Surface, Anatomical Site & Rate of Progression

#### Classification of Dental Caries

According to Virginity of Surface

Primary Caries

- Initial carious attack on a previously unaffected surface. Secondary / Recurrent Caries
- Occurs around existing restorations.

Associated with:

- Rough/overhanging margins.      Fractured restorations.
- Poor adaptation      Marginal leakage.

Residual Caries

- Caries left behind because excavation was incomplete.

According to Anatomical Site

1. Pit and Fissure Caries

- Most common.      Deep/narrow fissures retain plaque.
- Common in newly erupted teeth.      mutans is important in initiation.
- Usually prevented by appropriate sealant placement.

2. Smooth-Surface Caries

- Usually near gingival margin or beneath proximal contacts.
- Less favorable for plaque accumulation.
- Broad lesion with conical extension toward DEJ.

3. Linear Enamel Caries

- Associated with neonatal line and developmental/metabolic.





#### 4. Root-Surface Caries

- Common with gingival recession. More common in older individuals.
- Usually proximal/cervical.
- Rapid progression because cementum is less resistant.
- Often difficult to restore.

#### Classification According to Rate of Progression

##### Acute Caries

- Rapid progression. May affect many teeth.
- Light brown/gray. Soft, caseous dentin.
- Pulp involvement may occur early.

##### Chronic Caries

- Slow, long-standing. Dark brown. Leathery/firm dentin.
- Pain is less common. Secondary dentin may protect the pulp.

##### Arrested Caries

- No further progression. Lesion becomes hard/indurated.

Often associated with changes that eliminate plaque or food stagnation

#### Classification of Dental Caries: Extent, Tissue Involvement & Age

##### Classification According to Extent

##### Incipient Caries

- Early white-spot lesion. Surface still intact.
- Subsurface demineralization. May be remineralized.

##### Occult Caries

- Hidden lesion beneath relatively intact enamel.
- Enamel may appear relatively intact while dentinal destruction progresses.

##### Cavitated Caries

- Structural breakdown/cavity has developed.

Once caries reaches DEJ, dentinal spread becomes rapid





## Theories of Dental Caries & Histopathology of Enamel Caries

### *Theories of Dental Caries*

#### *1. Acidogenic / Chemico-Parasitic Theory – Miller*

*Bacteria + fermentable carbohydrate → acids → demineralization*

*Two major stages:*

- 1. Demineralization of inorganic tissues.*
- 2. Destruction/dissolution of the softened organic structure.*

*Critical plaque pH is around 5.5.*

#### *2. Proteolytic Theory*

*Proposes that bacteria initially attack the organic components of enamel.*

*Then:*

*Organic destruction → acids → mineral destruction → cavity formation*

*It helps explain some lesions that cannot be fully explained by the classical acidogenic theory.*

#### *3. Proteolysis-Chelation Theory*

*Suggests that:*

- Organic and inorganic components may be attacked simultaneously.*
- Proteolytic enzymes break the linkage between organic matrix and minerals.*
- Mineral salts and organic material are released.*

*There is limited experimental evidence for this theory*

### *Histopathology of Enamel Caries*

*A typical early enamel caries lesion has 4 zones, from the advancing front toward the surface:*

#### *1. Translucent Zone*

- Deepest/advancing front.*
- About 1% pore volume.*
- Increased porosity.*
- Mineral loss begins.*





## 2. Dark Zone

- Usually present. About 2–4% pore volume.
- Represents areas of remineralization/redeposition.
- Appears dark microscopically because very small pores trap air.

## 3. Body of Lesion

- Largest zone. Maximum demineralization.
- Approximately 5–25% pore volume.
- Mineral is replaced mainly by water and some organic material.
- Main zone responsible for the white appearance of early caries.

## 4. Surface Zone

- Approximately 30–40  $\mu\text{m}$ . Relatively intact.
- More resistant because of remineralization and mineral/fluoride availability.
- Explains how a lesion can exist beneath an apparently intact enamel surface.

## Histopathology of Dentin Caries

### Histopathology of Dentin Caries

Dentin caries is generally:

Cone-shaped  $\rightarrow$  broad base at DEJ  $\rightarrow$  apex toward pulp

Dentin caries progresses rapidly because:

- Dentin is less mineralized than enamel.
- It contains much more organic material.
- Dentinal tubules provide pathways for bacterial penetration.

### Defensive/Early Changes

From the pulp toward the carious lesion:

#### 1. Fatty Degeneration of Tomes' Fibers

- Degeneration of odontoblastic processes.
- Lipid droplets accumulate.

#### 2. Dentinal Sclerosis

- Mineral salts are deposited inside dentinal tubules.
- Tubules become less permeable.
- Protective response.





### 3. Zone of Dentinal Decalcification

- Mineral is removed by acids.

### 4. Zone of Bacterial Invasion

- Bacteria penetrate dentinal tubules.
- "Pioneer bacteria" may be found ahead of the main bacterial front.

### 5. Zone of Decomposed Dentin

- Extensive destruction by acids and bacterial/proteolytic activity.

## Advanced Dentinal Caries

### Liquefaction Foci

- Ovoid areas of destruction.
- Result from breakdown/coalescence of adjacent dentinal tubules.
- Filled with necrotic debris and microorganisms.

### Transverse Clefts

- Develop in necrotic dentin along planes of weakness.
- Run approximately perpendicular to the main dentinal tubules.
- Help carious dentin separate/peel away in thin layers.