DENTAL PLAQUE

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DEFINITION

DENTAL PLAQUE

“Is a specific but highly variable structural entity, resulting from sequential colonization of microorganisms on tooth surfaces, restorations & other parts of oral cavity, composed of salivary components like mucin, desquamated epithelial cells, debris & microorganisms, all embedded in extracellular gelatinous matrix.”

(\textit{WHO-1961})
DENTAL CALCULUS

is an adherent calcified or calcifying mass that forms on the surface of natural teeth & prosthesis.

MATERIA ALBA

is a deposit composed of aggregate of microorganisms, leucocytes & dead exfoliated epithelial cells, randomly organized & loosely adherent to the surfaces of the teeth, plaque & gingiva.
HISTORY OF DENTAL PLAQUE

- ANTONY VAN LEUWENHOEK - first one to describe dental plaque biofilms and their resistance.

- In 1899 G.V. BLACK coined the term "GELATINOUS MICROBIC PLAQUE".

- Waerhaug (1950) described bacterial plaque in the etiology of periodontal disease.

- Loe et al (1965) - plaque is main etiological agent in periodontal diseases.
CLASSIFICATION

- Coronal
- Marginal
- Fissural
- Supragingival
- Subgingival
- Tooth associated
- Unattached
- Tissue associated
SUPRA- GINGIVAL PLAQUE

• *Supragingival plaque* is found at or above the gingival margin.

• Supragingival plaque in direct contact with the gingival margin is referred to as *marginal plaque*.
Subgingival plaque is found below the gingival margin, between the tooth and gingival sulcular tissue.
<table>
<thead>
<tr>
<th>TOOTH ATTACHED</th>
<th>UNATTACHED</th>
<th>TISSUE ATTACHED</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gram positive – rods and cocci,</td>
<td>Gram negative rods, filaments, spirochetes</td>
<td>Both</td>
</tr>
<tr>
<td>Does not extend to JE</td>
<td>Extend to JE</td>
<td>Extend to JE</td>
</tr>
<tr>
<td>Calculus formation, root caries</td>
<td>Gingivitis</td>
<td>Gingivitis, periodontitis</td>
</tr>
<tr>
<td>May penetrate cementum</td>
<td>-</td>
<td>May penetrate epithelium and connective tissue</td>
</tr>
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COMPOSITION OF DENTAL PLAQUE

INTERCELLULAR MATRIX 20-30%

MICROORGANISM - 80%
INTERCELLULAR MATRIX

ORGANIC

INORGANIC
<table>
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<tr>
<th>ORGANIC MATRIX</th>
<th>INORGANIC MATRIX</th>
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<tr>
<td>Polysaccharide– produced by bacteria, e.g: dextran</td>
<td>Predominantly Ca, P- major Na, K, F - trace</td>
</tr>
<tr>
<td>Protein -albumin</td>
<td>source of inorganic material in supra-gingival plaque is primarily saliva.</td>
</tr>
<tr>
<td>Glycoprotein-from saliva</td>
<td>source of inorganic material in subgingival plaque is GCF</td>
</tr>
<tr>
<td>Lipid</td>
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</table>
One gram of plaque contains approximately $2 \times 10^{11}$ bacteria. 

(Socransky SS, 1953), (Schroeder, De Boever – 1970)

More than 500 distinct microbial species found in dental plaque – Moore 1994

Nonbacterial organisms are:
MYCOPLASMA
YEAST
PROTOZOA
VIRUSES
Socransky et al in 1998, 7 closely associated groups were recognized:
ACTINOMYCES SPECIES

V. Parvula
A. odontolyticus

S. Mitis
S. Oralis
S. Sanguis
Streptococcus sp.
S. gordonii
S. intermedius

PRIMARY COLONIZERS
SECONDARY COLONIZERS

P. Intermedia
P. Nigrescens
P. Micros
F. nucleatum
C. rectus
E. nodatum
C. showae

E. Corrodens
Capnocytophaga spp
A. actinomycetemcomitans
P. Gingivalis
B. Forsythus
T. denticola
SILVER COMPLEX

• HSV type 1
• EBSTEIN BARR VIRUS
• HUMAN CYTOMEGALO VIRUS
“Matrix enclosed bacterial populations adherent to each other and/or to surface or interfaces.”

(Costerton, 1978)

- Biofilms exist on any solid surface that is exposed to bacteria containing fluid.
EXOPOLYSACCHARIDES – the backbone of the biofilm

The bulk of the biofilm consists of the matrix, composed predominantly of water and aqueous solutes.

Function:
- Integrity of biofilm
- Prevents attack by harmful agents
- Assists in retention of extra cellular enzymes.
Quorum sensing in bacteria, "involves the regulation of expression of specific genes through the accumulation of signaling compounds that mediate intercellular communication."

(Prosser 1999)

This is a method of intercellular communication. Quorum sensing depends on cell density.

Once signaling compounds reach a threshold level, gene expression is activated.
QUORUM SENSING

Low Population density High

harmless type

aggressive type

Signal molecules

QS receptor

QS regulated genes

QS signal-receptor complex

Virulence genes up-regulated

e.g. biofilm formation, toxin production

24
Quorum sensing may give biofilms their distinct properties:

Expression of genes for antibiotic resistance at high cell densities may provide protection.

Has the potential to influence community structure, by encouraging the growth of beneficial species (to the biofilm) and discouraging the growth of competitors.

Alteration of physiological properties of bacteria in the community through quorum sensing.
Time for a short break....!!!!
FORMATION OF DENTAL PLAQUE
FORMATION OF PLAQUE

PELLICLE FORMATION ON TOOTH SURFACE

INITIAL ADHESION AND ATTACHMENT

COLONISATION AND PLAQUE MATURATION
I. FORMATION OF DENTAL PELLICLE

- **Acquired pellicle** may be defined as a homogenous, membranous, acellular film that covers the tooth surface and frequently form the interface between the tooth, the dental plaque and calculus. (SCHLUGER)

- A fully established pellicle is found within 30 min. Within 24 hr, the pellicle is around 0.1-0.8 µm in diameter.

- Derived from components of saliva and crevicular fluid as well as bacterial and host tissue cell products and food debris.
• Consists of numerous components, including glycoprotein (mucins), proline-rich proteins, phosphoproteins (e.g., statherin), histidine-rich proteins, enzymes (e.g., α-amylase), and other molecules that can function as adhesion sites for bacterial receptors.
FUNCTIONS OF DENTAL PELLICLE

- Protective barrier
- Lubrication
- Preventing tissue desiccation
- Substrate to which bacteria attaches
II. INITIAL ADHESION & ATTACHMENT OF BACTERIA

- We cannot conclude a single mechanism that dictates the adhesiveness of micro-organisms.

SCHEIE (1994)
A) TRANSPORT TO SURFACE

• The first stage involves the initial transport of the bacterium to the tooth surface.

• Random contacts may occur
  
  o Brownian motion (average displacement of 40 µm/hour)
  
  o Sedimentation of microorganisms,
  
  o Liquid flow
  
  o Active bacterial movement (chemotactic activity).
B) INITIAL ADHESION

- There is an initial, reversible adhesion of the bacterium.

- It is initiated by the interaction between the bacterium and the surface, from a certain distance (50 nm), through long-range and short-range forces, including van der Waals attractive forces and electrostatic repulsive forces.

- The total interaction energy, also called the total Gibbs energy ($G_{TOT}$).

- ($G_{TOT} = GA + GE$)
C) ATTACHMENT

- After initial adhesion, a firm anchorage between bacterium and surface will be established by specific interactions (covalent, ionic, or hydrogen bonding).

- The bonding between the bacteria & pellicle is mediated by specific extracellular components of organisms & complementary receptors on pellicle surface.
III. COLONIZATION & PLAQUE MATURATION

• The early colonizers (e.g., *streptococci* and *Actinomyces* species) use oxygen and lower the reduction-oxidation potential of the environment, which then favors the growth of anaerobic species.
• **Secondary colonizers** are the microorganisms that do not initially colonize clean tooth surfaces, including *Prevotella intermedia*, *Prevotella loescheii*, *Capnocytophaga spp.*, *Fusobacterium nucleatum*, and *Porphyromonas gingivalis*. 
Well characterized interaction include the coaggregation of:

- Fusobacterium nucleatum with all other human oral bacteria.
- Prevotella loescheii A. viscosus
- Capnocytophaga ochraceus A. viscosus
- Streptococci show intrageneric co-aggregation → bind to the nascent monolayer of already bound streptococci.

Later stages – coaggregation between different Gram negative species seen – F. nucleatum & P. gingivalis or T. denticola.
CORN COB formation – streptococci adhere to filaments of Bacterionema species or F.nucleatum.
COAGGREGATION BRIDGES

- A co-aggregation bridge is formed when the common partner bears two or more types of coaggregation mediators.

- These mediators can be various types of polysaccharides or various adhesin or combination of two
DENTAL PLAQUE FORMATION – RELATION TO TIME
Bacteria adhere to pellicle, and pellicle coats the enamel.

Gram positive rods and cocci are laid down in the first hour.
Bacteria multiply and form mini-colonies in layers upon the pellicle.

The bacteria adhere and increase in mass and thickness.
As the plaque thickens at the cervical area, the deeper layers incorporate more *filaments* and *fusiforms*, eventually turning *gram negative*. The coronal plaque is a more simple early arrangement of rods and cocci. Bleeding on probing and erythema can be seen.
As the plaque continues to mature, *vibrio*, *spirochetes*, and *white blood cells* appear. The plaque becomes more gram negative and anaerobic in the deeper layers.

The signs of inflammation are more pronounced.
• **Vibrio and spirochetes** continue to multiply.
• The bacteria become
  ✓ Highly organized
  ✓ Filamentous
  ✓ Perpendicular to the tooth surface
• The signs of inflamed gums are obvious
A disclosing agent is a preparation in liquid, tablet or lozenge form which contains a dye or other contouring agent.

- Iodine preparation
- Bismarck brown
- Erythrosine
- Fast green
- Basic fucshin
NON-SPECIFIC PLAQUE HYPOTHESIS

SPECIFIC PLAQUE HYPOTHESIS

ECOLOGICAL PLAQUE HYPOTHESIS
NON-SPECIFIC PLAQUE HYPOTHESIS

- (Theilade 1976) held that the entire bacterial flora in plaque played a role in periodontal destruction rather than specific bacteria.

- The nonspecific plaque hypothesis maintains that periodontal disease results from the “elaboration of noxious products by the entire plaque flora.”

- Thus it lead to concept that control of periodontal disease depends on control of the amount of plaque accumulation.
Specific plaque hypothesis- Walter Loesche 1979

states that only certain plaque is pathogenic, and its pathogenicity depends on the presence of or increase in specific microorganisms.
Plaque harboring specific bacterial pathogens results in periodontal disease.

- *A. actinomycetemcomitans* is a pathogen in aggressive periodontitis.
ECOLOGICAL PLAQUE HYPOTHESIS

• A change in a key environmental factor (or factors) will trigger a shift in the balance of the resident plaque microflora, and this might predispose a site to disease. (PD Marsh 1994)

• This hypothesis is based on the theory that the unique local microenvironment influences the composition of the oral microflora.
Reduced plaque → Reduced inflammation → Low GCF flow → Gram-positive flora

Stress → Environmental change → Ecological shift → Gingivitis

Increased plaque → Increased Inflammation → High GCF flow, bleeding, raised pH & temp, low Eh → Gram-negative anaerobes
In the 1870s, Robert Koch postulated the criteria by which an organism can be judged to be causative agent in human infections.
KOCH POSTULATES

Pathogen must be **routinely isolated** from the diseased individuals.

Must be **grown in pure culture** in the laboratory.

Must produce a **similar disease** when inoculated into susceptible lab animals.

Must be **recovered from lesions** in a diseased laboratory animals.
SIGMUND SOCRANSKY (1978) proposed criteria by which periodontal microorganisms may be judged to be potential pathogens.

- **Association**
- **Elimination**
- **Host response**

**Virulence factors**

Must be capable of causing disease in experimental animal models.
Microbial shift from health to disease

1) Gm + ve to Gm –ve
2) Cocci to rods to spirochaetes
3) Non- motile to motile bacteria
4) Facultative anaerobes to obligate anaerobes
REFERENCES

• Clinical Periodontology - Carranza (9th & 10th edn)

• Clinical Periodontology - Jan Lindhe, Thorklid Karring, Niklaus P Lang

• Clinical Periodontology: Listgarten

• Are dental diseases examples of ecological catastrophes? - P. D. Marsh - Microbiology (2003), 149, 279–294


1. Definition of dental plaque given by:
   a. WHO 1974
   b. WHO 1961
   c. WHO 1984
   d. WHO 1955
   Ans. B

2. Marginal plaque is a type of:
   a. Subgingival plaque
   b. Supragingival plaque
   c. both
   d. None of the above
   Ans. B
3. Subgingival plaque is rich in-
   a. GM +ve microbes
   b. GM-ve microorganisms
   c. Fungi
   d. none
   **Ans- b**

4. Microbial complexes were given by-
   a. Socransky
   b. Loe
   c. Pierre Fauchard
   d. Glickman
   **Ans- a**
5. RED complex contains which one?
   a. A. actinomycetemcomitans
   b. P. gingivalis
   c. Streptococcus sp.
   d. Vellionella sp.
   
   **Ans-b**

6. Which complex is associated with bleeding on probing?
   a. RED
   b. BLUE
   c. PURPLE
   d. GREEN
   
   **Ans- a**
7. Specific plaque hypothesis given by-
   a. Walter Loesche
   b. Thelaide
   c. Marsh
   d. None
   Ans-a

8. Transport of bacteria to the tooth surface occurs by-
   a. Brownian motion
   b. Liquid flow
   c. sedimentation
   d. All of the above
   Ans- d
9. Which is a primary colonizer?
   a. P. gingivalis
   b. T. denticola
   c. Streptococcus
   d. None of the above
   Ans-c

10. Which is an example of bacterial interactions–
   a. Quorum sensing
   b. Corncob formation
   c. Test-tube brush formations
   d. All of the above
   Ans- d
THANK YOU
FOR LISTENING

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