THIS IS JUST A SUPPLEMENTARY MATERIAL TO HELP IN YOUR EXAMINATIONS; IT SHOULD NOT REPLACE YOUR TEXT BOOKS. KINDLY GO THROUGH YOUR TEXTBOOKS FIRST FOR BETTER UNDERSTANDING OF THESE NOTES.

DENTAL PLAQUE

DEFINITIONS:

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."

W.H.O ,1961

Dental Plaque can be defined as

soft deposits that form the biofilm adhering to the tooth surface or other hard surfaces in the oral cavity, including removable & fixed restorations"

Bowen, 1976

TYPES:

Dental plaque is broadly classified based on its position on the tooth surface as

- Supra gingival plaque
- Subgingival plaque
    - Attached subgingival plaque
    - Unattached subgingival plaque or
    - Tooth associated plaque
    - Tissue associated plaque.
- Marginal plaque

Supra gingival plaque

It is found at or above the gingival margin. The supra gingival plaque i.e. is in direct contact with the gingival margin is referred to as marginal plaque.

Subgingival plaque

It is found below the gingival margin between the tooth and the gingival sulcular tissue.

Attached subgingival plaque:-

In the gingival sulcus and periodontal pocket, there is a zone of plaque bacteria which is attached to the tooth surface. The attached component is apparently continuous with the supragingival plaque and usually extends near the apex of the gingival sulcus or periodontal pocket in chronic periodontitis.

Unattached Subgingival plaque:-

Another discernible area within the subgingival plaque is not directly attached to the tooth surface. The unattached component of the subgingival plaque in chronic periodontitis extends from the gingival margin to the junctional epithelium.

Thus different regions of plaque are significant to different processes associated with diseases of the teeth and periodontium.For ex.

- Marginal plaque is of prime importance on the development of gingivitis.
- Supragingival plaque and tooth associated subgingival plaque are critical in calculus formation and root caries.
- Whereas tissue associated subgingival plaque is important in the soft tissue destruction characteristic of different forms of periodontitis.

COMPOSITION OF DENTAL PLAQUE:-

Structural composition

- Plaque contains 80% of water and 20% of organis and inorganic solid.
- Bacteria constitutes approximately 70 to 80% of the solid material, and the rest is intercellular matrix derived from saliva, GCF and bacterial product.
- Organic constituents of the matrix include
    - Polysaccharides: produced by the Bacteria of which Dextran is the predominant one.

- Proteins : obtained from the crevicular FLUID.
- Glycoproteins : from saliva which is an important component of the pellicle that initially coats the clean tooth surface.
- Lipid : onsist of debris from the membrane of disrupted bacterial and host cells and possibly found debris.

Inorganic component:

- It is primarily calcium and phosphorus, with trace amounts of other mineral such as sodium, potassium and fluoride.
- The source of inorganic component of supragingival plaque is mainly saliva and inorganic component of subgingival plaque source is cervicular Fluid.
- Only the fluoride component of plaque is largely derived from external sources such as fluoridated tooth pastes and Rinses.

Bacterial composition of the Plaque:

- Dental plaque is composed primarily of microorganism. One gram of plaque (wet weight) contains approsimately 2 x10”bacteria. It has been estimated that more than 325 different bacterial species may be found in the plaque (Moore WEC)
- Non bacterial microorganism that are found in plaque include mycoplasma species, yeasts, protozoa and viruses.

Bacterial composition of supra gingival plaque:-

Gram positive Cocci and shot Rods predominate at the tooth surface, whereas gram negative Rods filaments as well spirochetes predominate in the outer surface of the mature plaque mass.

Bacterial composition of Subgingival plaque:

- Attached (tooth associated) Plaque:- It is characterized by gram positive rods and cocci including bacteria such as
    - Streptococcus mitis
    - S. Sanguis
    - Actinomyces Viscosus
    - A Naeslundii
    - Eubacterium species.

Unattached (tissue Associated) Plaque:

It contains primarily gram negative rods and Cocci as well as large numbers of filaments, flagellated rods and spirochetes.

FORMATION OF DENTAL PLAQUE

The process of plaque formation can be divided into three major phases

- Formation of the pellicle
- Initial adhesion and attachment of Bacteria
- Colonization and Plaque formation

Pellicle formation:

- The acquired pellicle is tenacious membraneous layer that is amorphous, acellular and organic, that forms on the exposed tooth surfaces, restoration ass well as over dental calculus
- Its thickness varies from 0.1 to 0.8 mm
- All surface of the oral cavity both hard and soft tissues are coated with a pellicle.
- This pellicle consists of numerous components like glycoproteins, proline rich proteins,phosphoproteins, histidine rich proteins, enzymes like alfa amylase
- Pellicle forms by selective adsorption of environmental macromolecules

The mechanisms involved in pellicle formation are

1. Electrostatic
2. Van der waals
3. Hydrostatic forces

Initial adhesion and attachment of Bacteria

- Rapid colonization by pioneer species (Gram +ve cocci and rods) :- 1-2 days
- *S. sanguis, S. oralis, S. mitis, A. viscosus can adhere to* pellicle by specificity
- Resisting shear force (saliva) and electrostatic repulsion
- The initial bacteria are called “pioneer bacteria”.

It is a 4-staged sequence:

Phase 1. Transport to the surface:

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

Random contacts may occur by

- Brownian motion
- Sedimentation of the microorganisms
- Liquid flow
- Active bacterium movement

Phase 2. Initial adhesion:

- The second stage results in an initial reversible adhesion of microorganisms, Initiated by the interaction between the bacterium and surface through long and short range forces including van der waals attractive forces, electrostatic repulsive forces
- A group of short range forces ie **hydrogen bonding, ion pair formation, and steric interaction** dominates the adhesive interaction and determines the strength of adhesion

Phase 3. Attachment:

- After initial adhesion, a firm anchorage between bacterium and surface will be established by specific interaction [covalent, ionic or hydrogen bonding]
- On rough surface, bacteria are better protected against shear forces so that a change from reversible to irreversible bonding occurs more easily and more frequenty.
- The bonding between bacteria and pellicle is mediated by specific extracellular proteinaceous components[adhesions] of the organisms and complementary receptors on the surface and is species specific.

For eg. Streptococcus the principal early colonizer, bind to acidic proline-rich- proteins and other receptors in the pellicle, such as alfa amylase and sialic acid

Colonization and plaque maturation

- Secondary colonizers are the microorganisms that do not initially colonize clean tooth surface.
- These microorganisms adhere to cells of bacteria already in the plaque mass.
- Extensive lab studies have documented the ability of different species and genera of plaque microorganisms to adhere to one another, a process known as coaggregation.

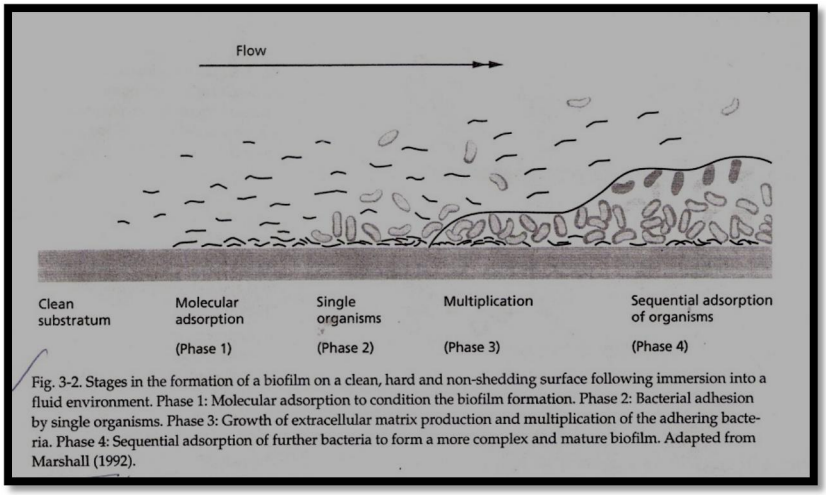


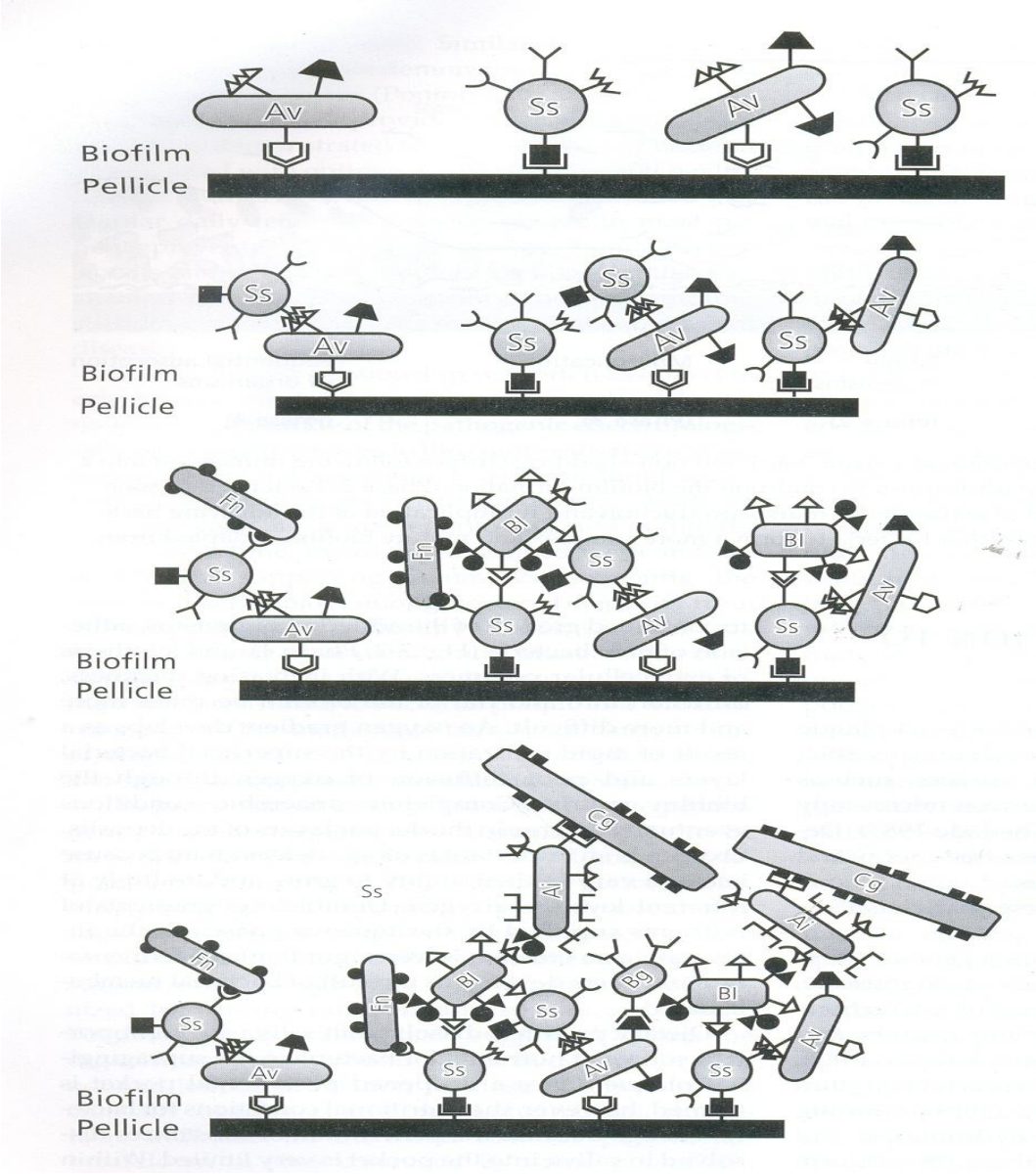
Fig. 3-2. Stages in the formation of a biofilm on a clean, hard and non-shedding surface following immersion into a fluid environment. Phase 1: Molecular adsorption to condition the biofilm formation. Phase 2: Bacterial adhesion by single organisms. Phase 3: Growth of extracellular matrix production and multiplication of the adhering bacteria. Phase 4: Sequential adsorption of further bacteria to form a more complex and mature biofilm. Adapted from Marshall (1992).

Coaggregation:

Occurs primarily through the highly specific stereochemical interactions of protein & carbohydrate molecules located on the bacterial cell surfaces, in addition to the less specific interactions resulting from hydrophobic, electrostatic & van der Waals forces.

Kolenbrander PE et al 1987

Eg: "Corn cob": streptococci adhere to filaments of B.matruchotii or Actinomyces species.



PLAQUE AS A BIOFILM

- Bacteria in a biofilm are grouped in microcolonies surrounded by an enveloping intermicrobial matrix
- The matrix is penetrated by fluid channels that conduct the flow of nutrients, waste products, enzymes, metabolites, and oxygen.
- These microcolonies have micro environments with differing pH's, nutrient availability, and oxygen concentrations.
- The bacteria in a biofilm communicate with each other by sending out chemical signals.

- These chemical signals trigger the bacteria to produce potentially harmful proteins and enzymes
- Microorganisms in biofilm are resistant to antibiotics, antimicrobials and host response.

MICROBIAL COMPLEXES IN PLAQUE BIOFILM

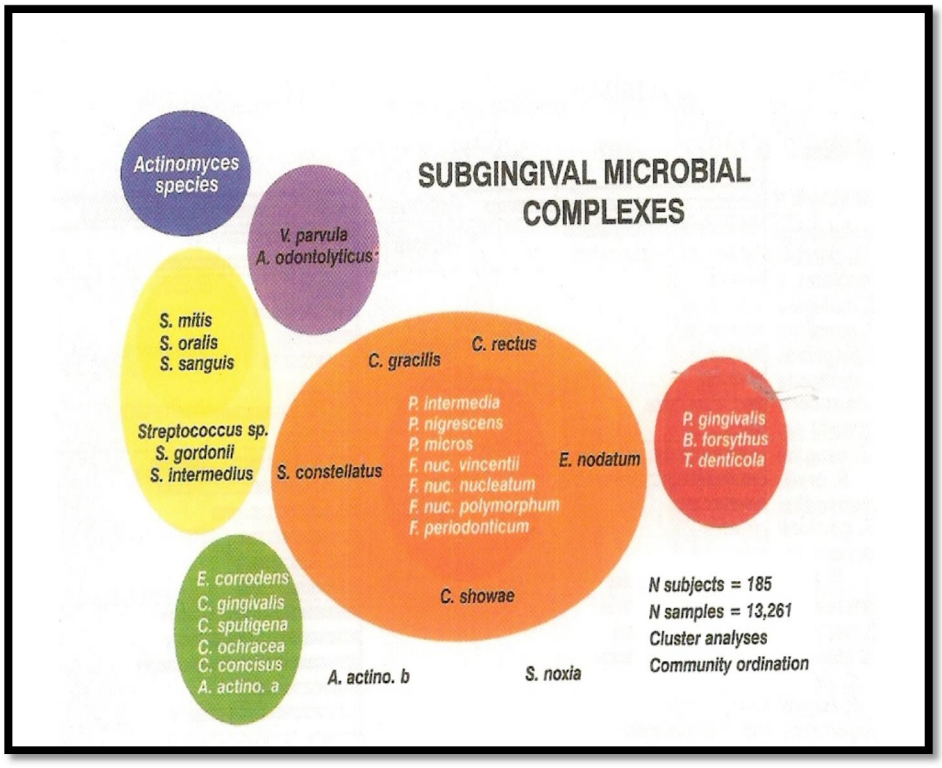
Transition from a healthy oral environment to gingivitis and to periodontal disease is triggered by a specific 'set' or 'complex' of bacterial species

Early colonizers:

- Independent or defined complexes (Actinomyces naeslundii, A. viscosus)
- Yellow complex (streptococcus spp)
- Purple complex (A. odontolyticus)

Secondary and Tertiary colonizers:

- Green (E.corrodens, A.actinomycetemcomitans serotype a & capnocytophaga species)
- Orange (fusobacterium, prevotella & campylobacter spp)
- Red complex (P. gingivalis, T. forsythia, & T. denticola)



Non Specific Plaque Hypothesis-

- Given in 1976 by **Walter Loesche**
- According to this theory, when only small amounts of plaque are present, the noxious products are neutralized by the host.
- Similarly, large amounts of plaque would produce large amounts of noxious products, which would essentially overwhelm the host's defenses.
- The current standard treatment of periodontitis by debridement (nonsurgical or surgical) and oral hygiene measures still focuses on the removal of plaque and its products and is founded in the nonspecific plaque hypothesis.

Specific Plaque Hypothesis

- The specific plaque hypothesis was put forth by Walter Loesche in 1979.
- The specific plaque hypothesis states that only certain bacteria are pathogenic and its pathogenicty depends on the presence of or increase in specific microorganisms.
- This concept predicts that plaque harbouring specific bacterial pathogens results in periodontal disease because these organisms produce substances that mediate the destruction of host tissues.
- The acceptance of this concept was spurred by the recognition of A.a comitans as the pathogenic agent of Localised Aggressive Periodontitis.
- This was possible because of improved laboratory procedures to sample subgingival plaque.

Ecological Plaque Hypothesis- (MARSH 1991)

- In this hypothesis, it is proposed that 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
- Disease results from shifts in the balance of the resident plaque microflora.
- Potentially pathogenic bacteria can be present in health, but at levels that are not clinically relevant.
- Disease could be controlled not only by targeting putative pathogens but also by interfering with the factors responsible for driving the deleterious shifts in the microflora.

MICROORGNISM ASSOCIATED WITH PERIODONTAL HEALTH

- The bacteria associated with periodontal species are primarily gram +ve facultative species, and member of these genera are *Streptococcus* and *Actinomyces (S.sanguis, S.mitis, A.viscosus & A.naeslundii).*
- Benefecial species are *S.sanguis, Veilonella parvula, C.orchracea.*
- They function by preventing colonization of pathogenic microorganisms.
  Eg. *S.sanguis* produces H_2O_2 which is lethal to the cell of *A.a.*

CONCLUSION

The view of plaque and its constituents microorganisms have shifted from specific plaque hypothesis to a non-specific plaque hypothesis and back again to a theory of specific periodontal pathogens to plaque. Recently dental researchers have begun to view plaque as a biofilm. The nature of a biofilm helps explain why periodontal diseases have been so difficult to prevent and treat. An improved understanding of biofilm will lead to new strategies for management of these widespread diseases.