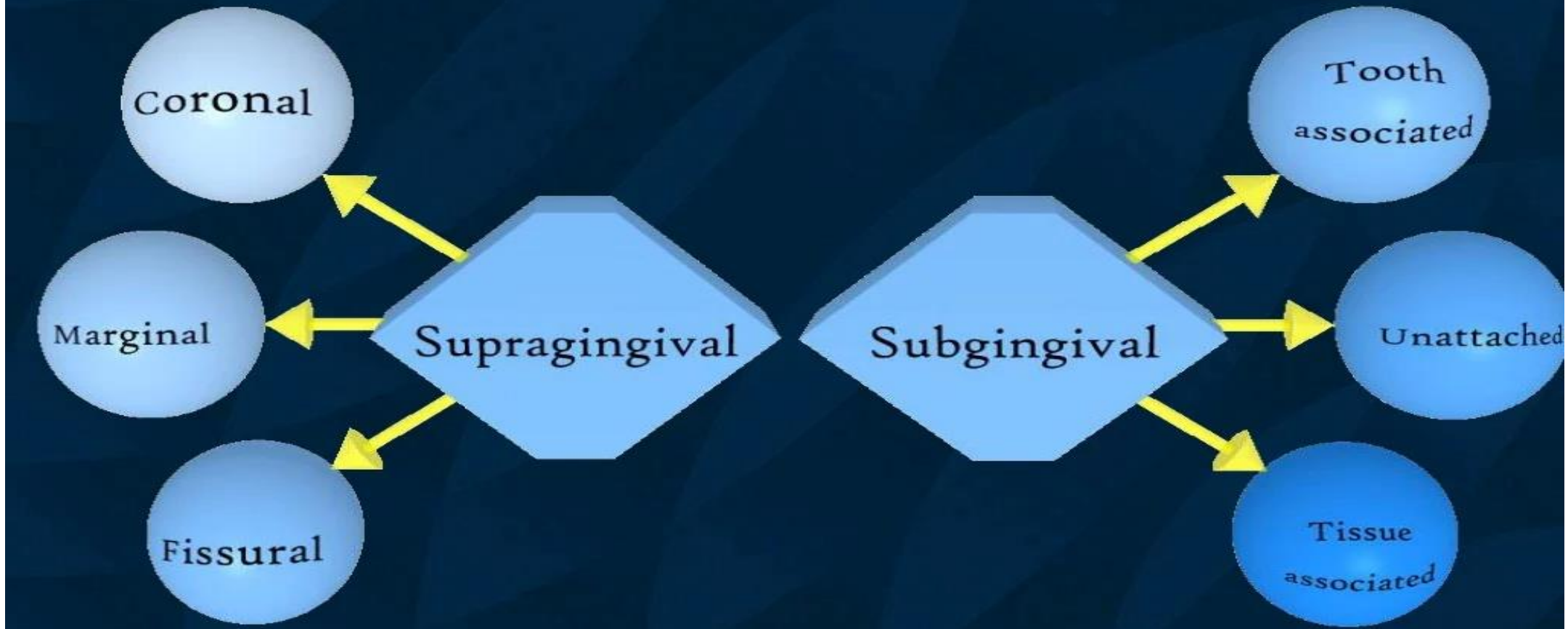
Dental plaque : is defined clinically as a structured, resilient, yellow-grayish substance that adheres tenaciously to the intraoral hard surfaces, including removable and fixed restorations.

The tough extracellular matrix makes it impossible to remove plaque by rinsing or with the use of sprays.

Materia alba refers to soft accumulations of bacteria, food matter, and tissue cells that
lack the organized structure of dental plaque and that are easily displaced with a water spray.

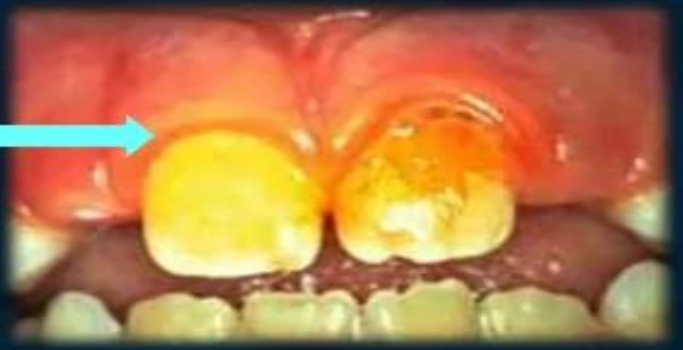
Calculus is a hard deposit that forms via the mineralization of dental plaque and that is generally covered
by a layer of unmineralized plaque

CLASSIFICATION

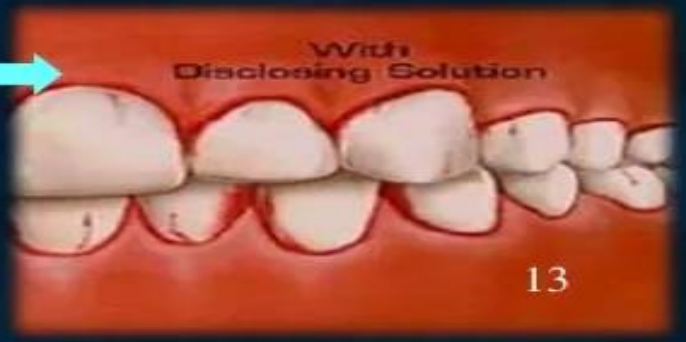


SUPRA- GINGIVAL PLAQUE

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

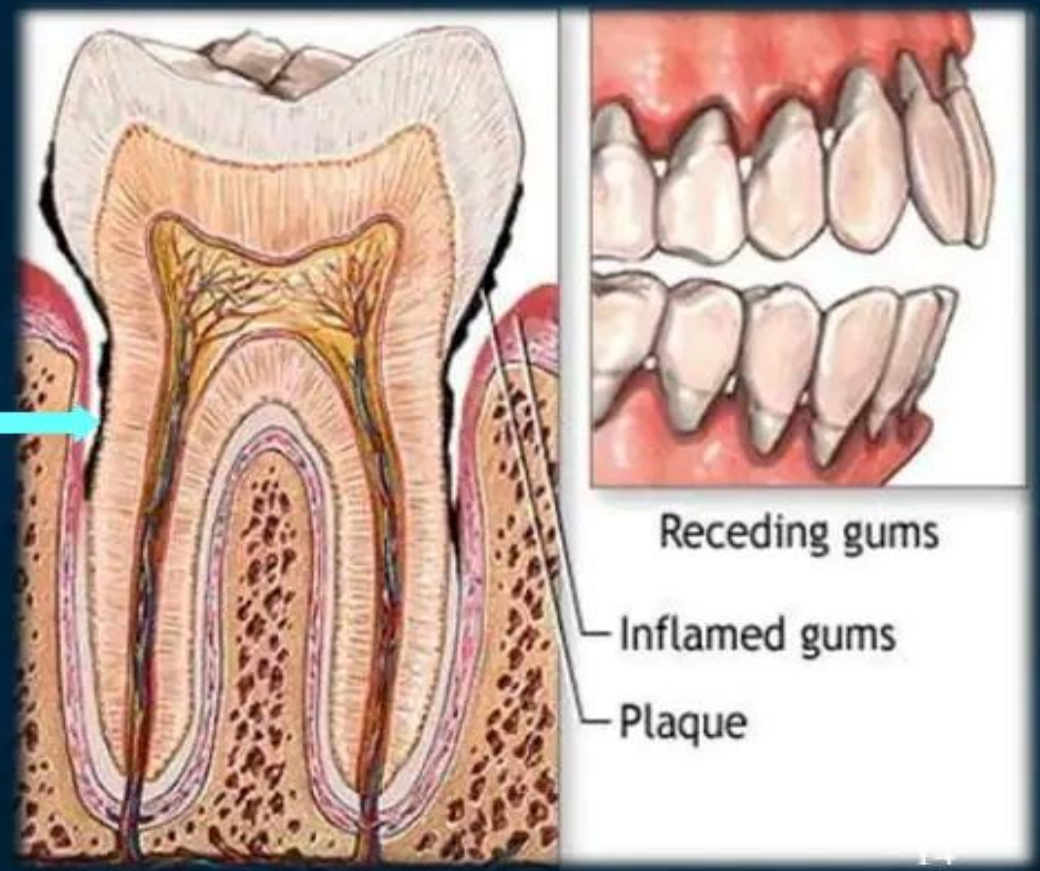


- Supragingival plaque in direct contact with the gingival margin is referred to as ***marginal plaque***

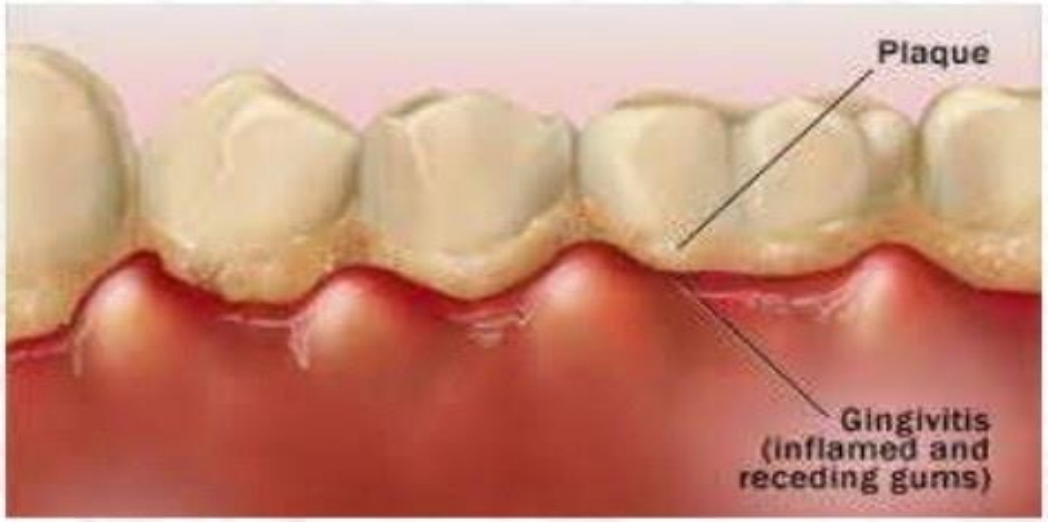


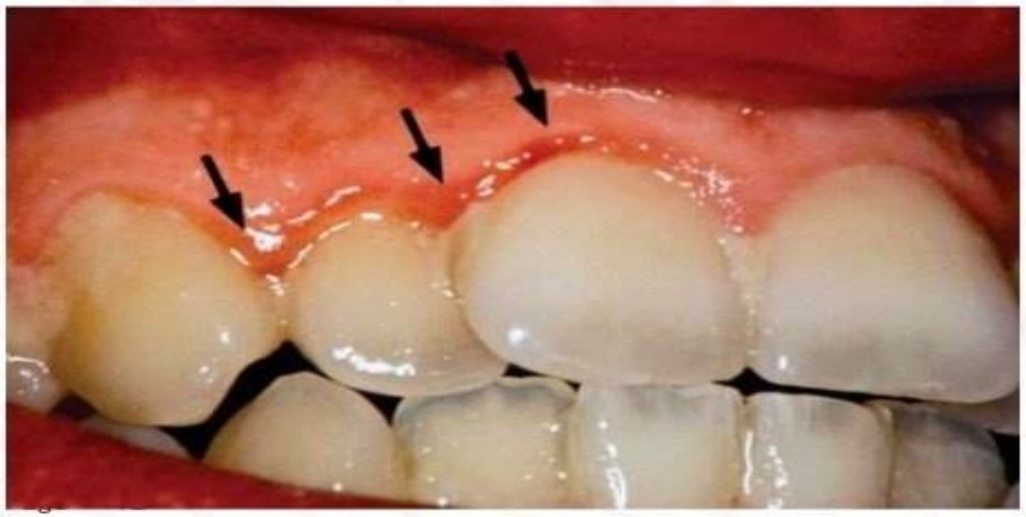
SUBGINGIVAL PLAQUE

- ***Sub gingival plaque*** is found below the gingival margin, between the tooth and gingival sulcular tissue.



SUPRAGINGIVAL PLAQUE

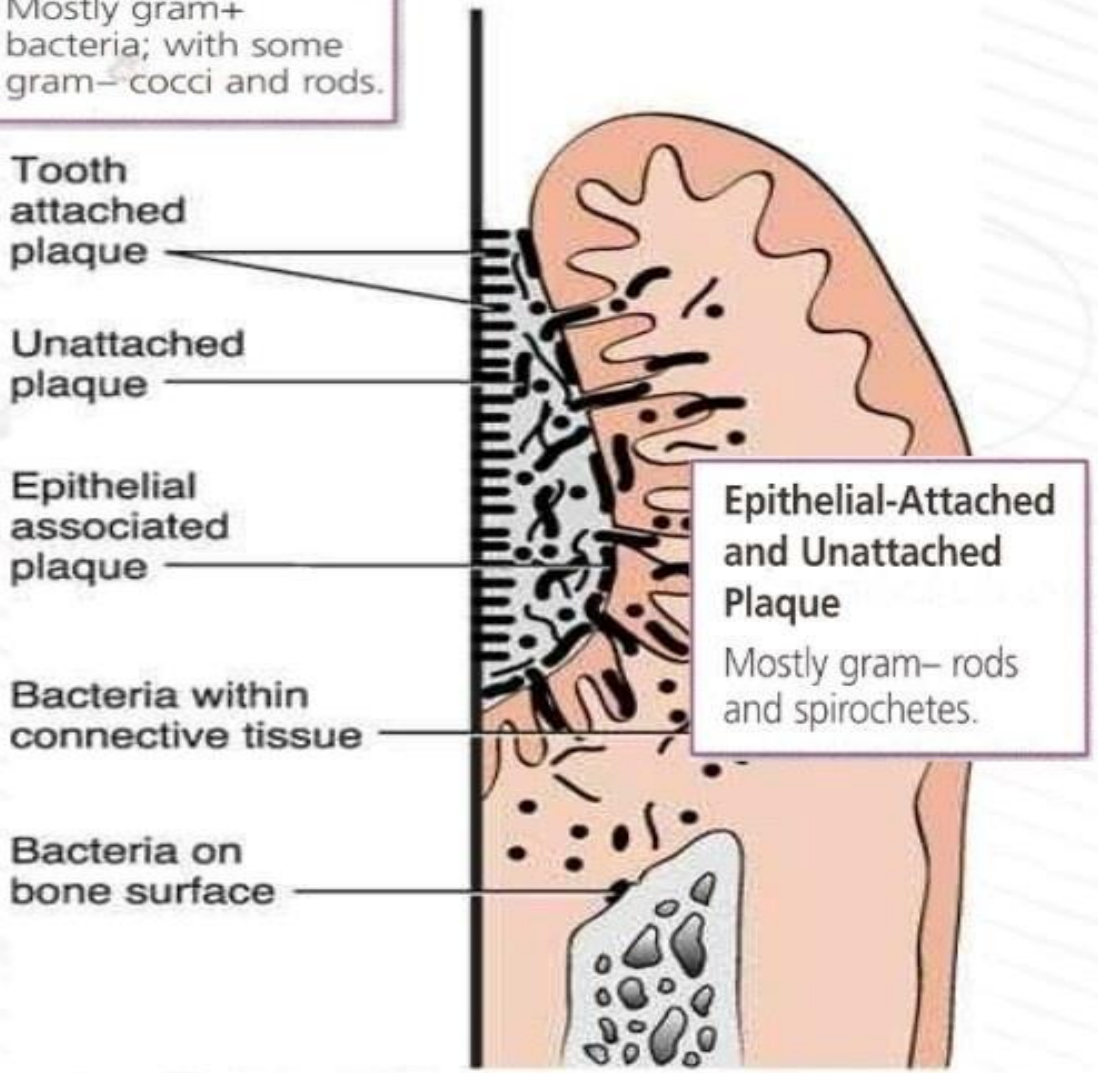




SUBGINGIVAL PLAQUE

Tooth-Attached Plaque

Mostly gram+ bacteria; with some gram– cocci and rods.



Tooth attached	Unattached	Tissue Attached
Gram positive – rods and cocci,	Gram negative rods, filaments, spirochetes	Gram variable
Does not extend to JE	Extend to JE	Extend to JE
Calculus formation, root caries	Gingivitis	Gingivitis, periodontitis
May penetrate cementum	-	May penetrate epithelium and connective tissue

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

Supragingival Plaque

Found at or above the gingival margin.

Demonstrates a stratified organization of a multilayered accumulation of bacteria

Gram-positive cocci and short rods predominate at the tooth surface,

Gram-negative rods and filaments, as well as spirochetes, predominate in the outer surface of the mature plaque mass.

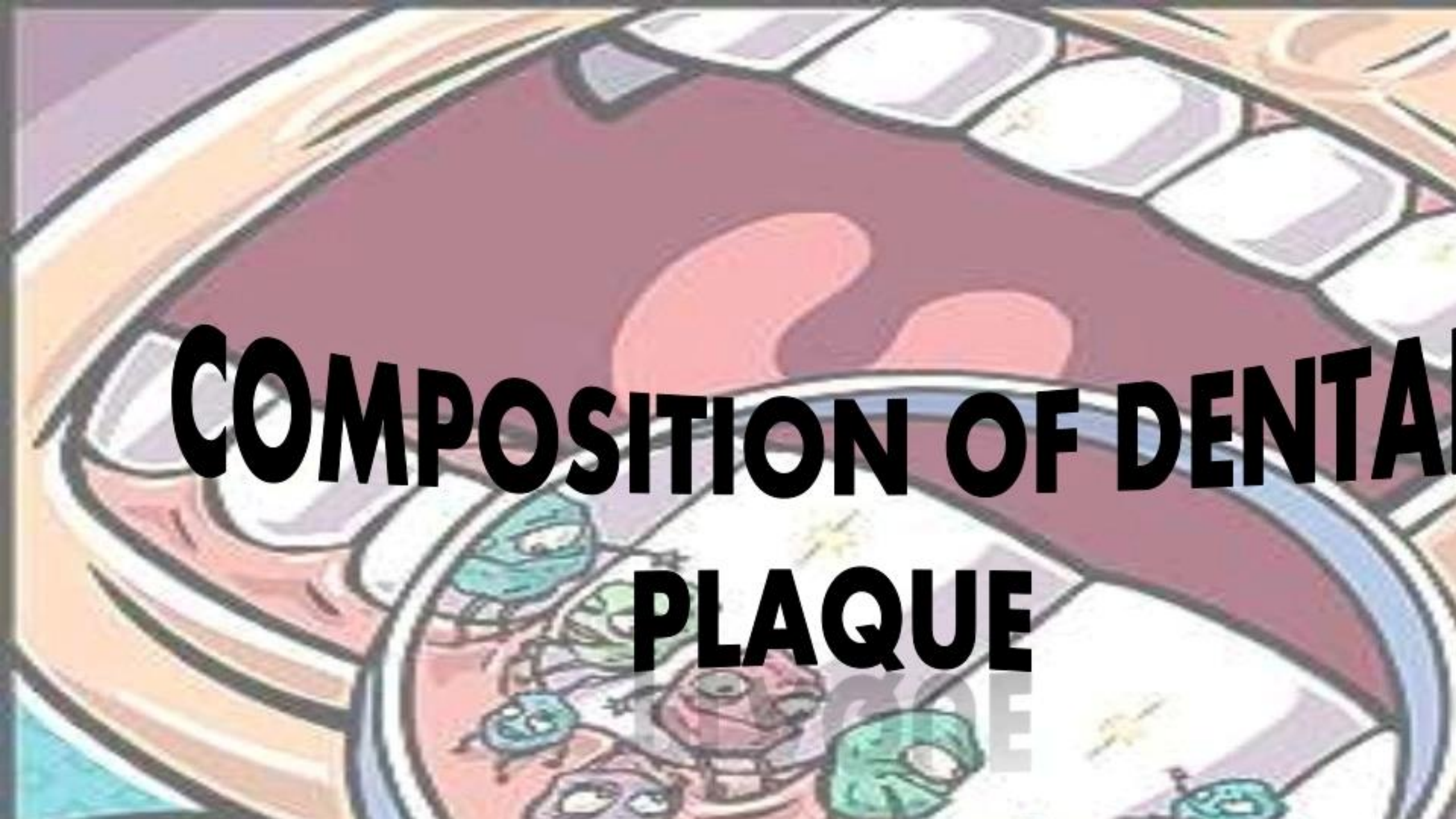
Subgingival Plaque

Found below the gingival margin, between the tooth and the gingival pocket epithelium.

The composition of the subgingival plaque depends on the pocket depth.

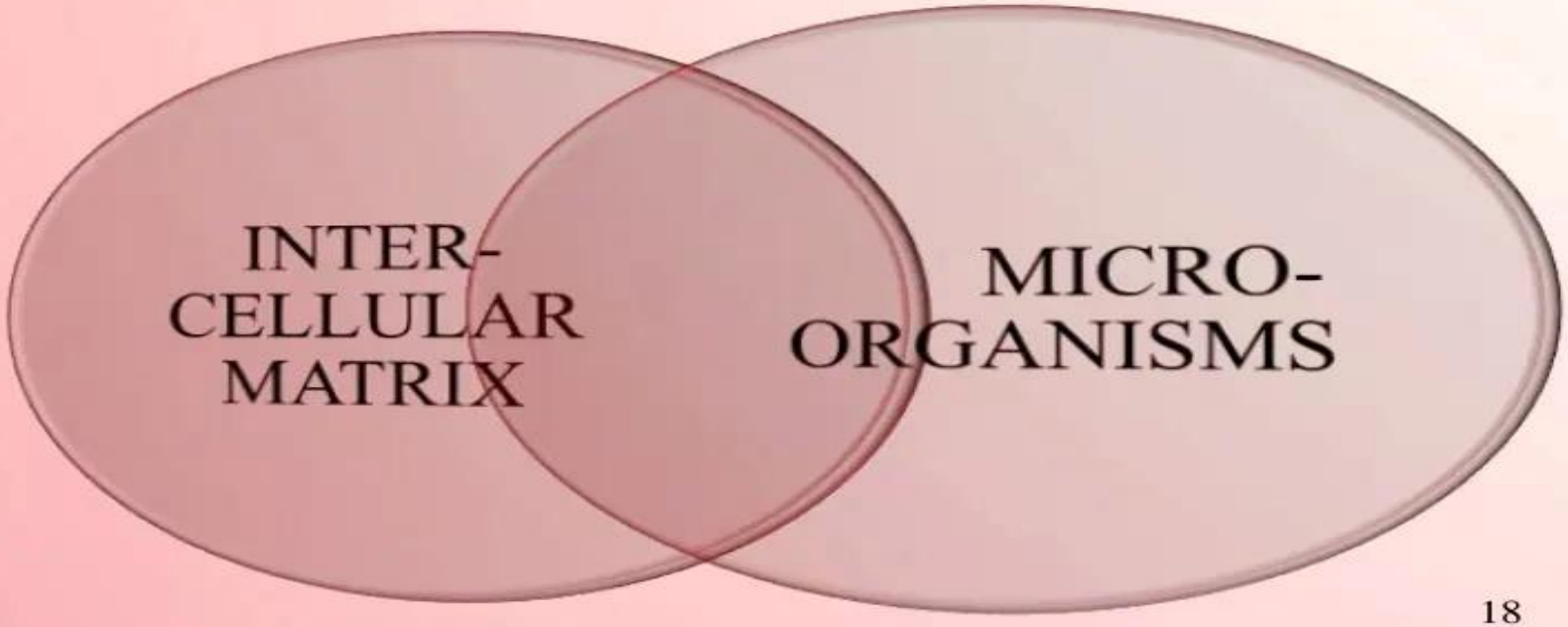
The subgingival microbiota differs in composition primarily because of the local availability of blood products and an anaerobic environment.

The apical part is more dominated by spirochetes, cocci and rods, whereas in the coronal part more filaments are observed.

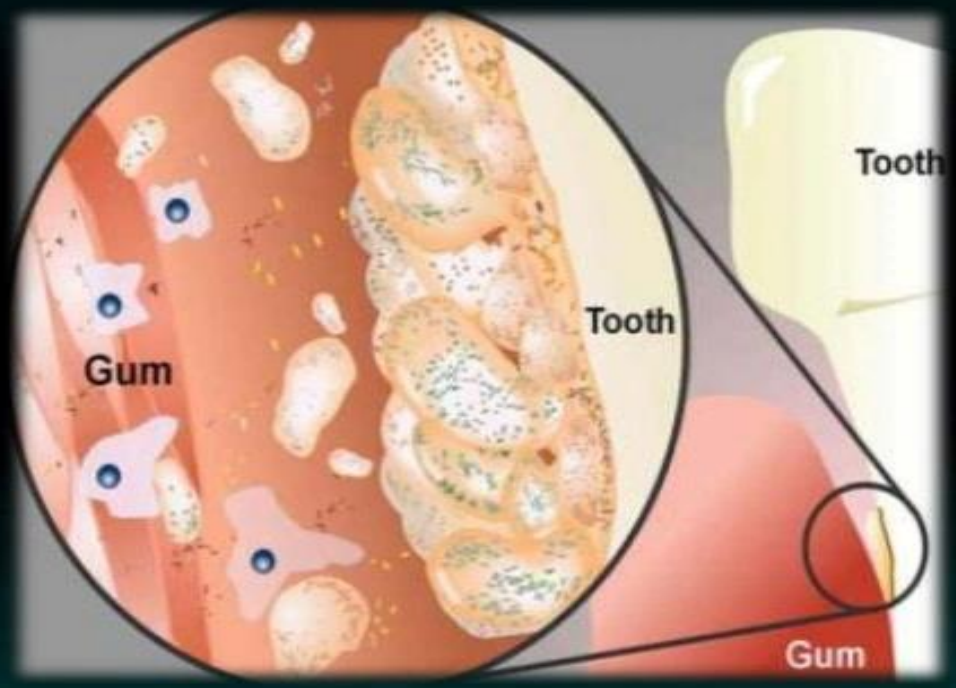

COMPOSITION OF DENTA
PLAQUE

COMPOSITION OF DENTAL PLAQUE

- Dental plaque is composed primarily of microorganisms.
- These organisms exist within an intercellular matrix.



BIOFILM



The term biofilm describes the relatively indefinable microbial community associated with a tooth surface or any other hard, non-shedding material.

(Wilderer & Charaklis 1989)

- Biofilm consists of one or more communities of micro-organisms embedded in glycocalyx , that are attached to solid surfaces.

(Costerton et al 1994)

EXOPOLYSACCHARIDES – the backbone of the biofilm

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

Maintaining the **integrity & protecting microbial cells** within the biofilm by preventing desiccation and attack by harmful agents.

They **bind essential nutrients** such as cations to create a nutritionally rich environment favouring specific micro-organisms.

Act as a **buffer and assist** in retaining extracellular enzymes (and their substrates), enhancing substrate utilization by bacterial cells.

Composition of dental plaque

PLAQUE

SOLIDS 20-30%

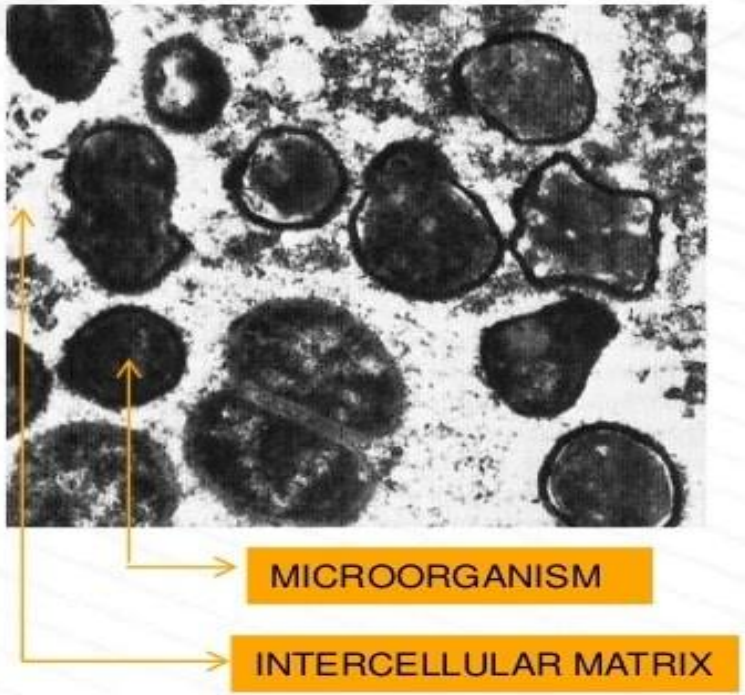
WATER 70-80%

Micro-organisms 70%

- Bacterial
- Non-bacterial

Intercellular Matrix 20%-30%

- Organic material
- Inorganic materials



INTERCELLULAR MATRIX

- This contains a few host cells such as epithelial cells , macrophages & leukocytes.
- Forms 20%-30 % of plaque mass.

ORGANIC MATERIAL

- Polysaccharides (30%) produced by bacteria.
  Dextran
- Protein (30%) –Albumin (originating from GCF)
- Glycoproteins from saliva.
- Lipid (15%) consists of debris from disrupted bacteria,
  host cells & food debris

INORGANIC MATERIAL

- Predominantly – calcium, sodium, phosphorous, potassium & fluorides (in traces)
- Source of inorganic material in supragingival plaque is primarily **saliva**
- source of inorganic material in sub gingival plaque is **GCF**

BACTERIAL PORTION

- 70 to 80 % of total solid plaque volume.
- 1 gm of plaque contains approximately 2 X 10^{11} bacteria.

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

Bacteria	Facultative	Anaerobic
Gram +ve	■Strep.mutans ■Strep.sanguis ■A.viscosus	
Gram -ve	■A.actinomycetemcomitans ■Capnocytophypa sp. ■Ekinella corrodens	■P.Gingivalis ■F.nucleatum ■P.intermedia ■B.forsythus ■C.rectus
Spirochetes		■ T.denticola

NON BACTERIAL PORTION

MICROSCOPIC STRUCTURE

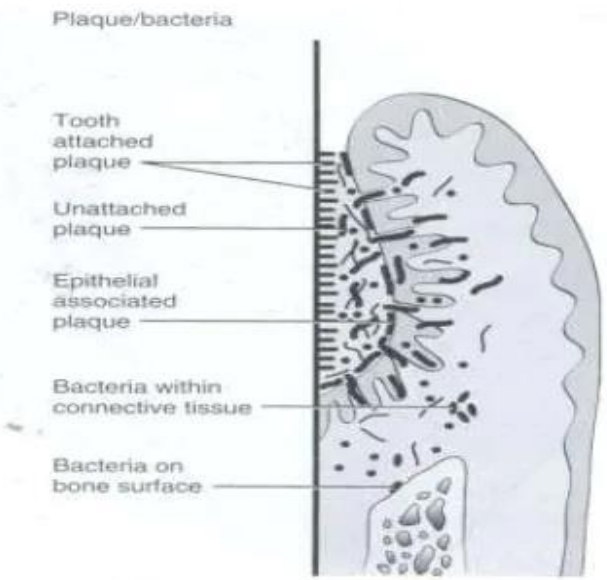
SUPRAGINGIVAL PLAQUE

- Typically demonstrates a stratified organization of the bacterial morphotypes.
- Gram-positive cocci and short rods predominate at the tooth surface
- Gram-negative rods and filaments ,spirochetes predominate in the outer surface of the mature plaque mass.
- *Supra gingival plaque* can have a structured architecture polymer containing channel or pores have been observed that link the plaque/oral environment interface to the tooth surface *(Wood et al 2000,Auschillet al 2001,Zaura Arite et al 2001)*

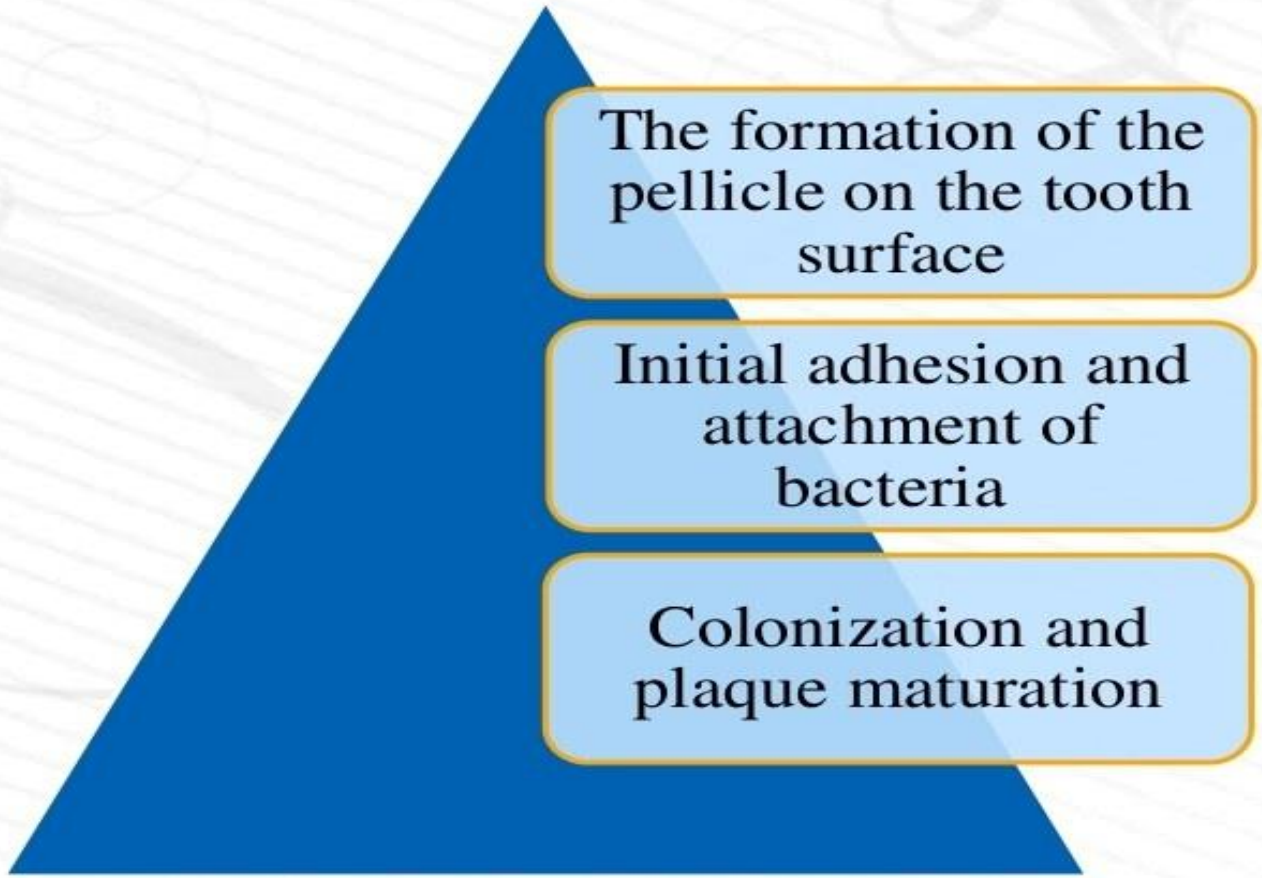
SUBGINGIVAL PLAQUE

- Between sub gingival plaque and the tooth an electron dense organic material is interposed , termed as cuticle.
- Gingival crevicular fluid, -contains many substances that the bacteria may use as nutrients
- Host inflammatory cells and mediators have influence on the establishment and growth of bacteria in this region.

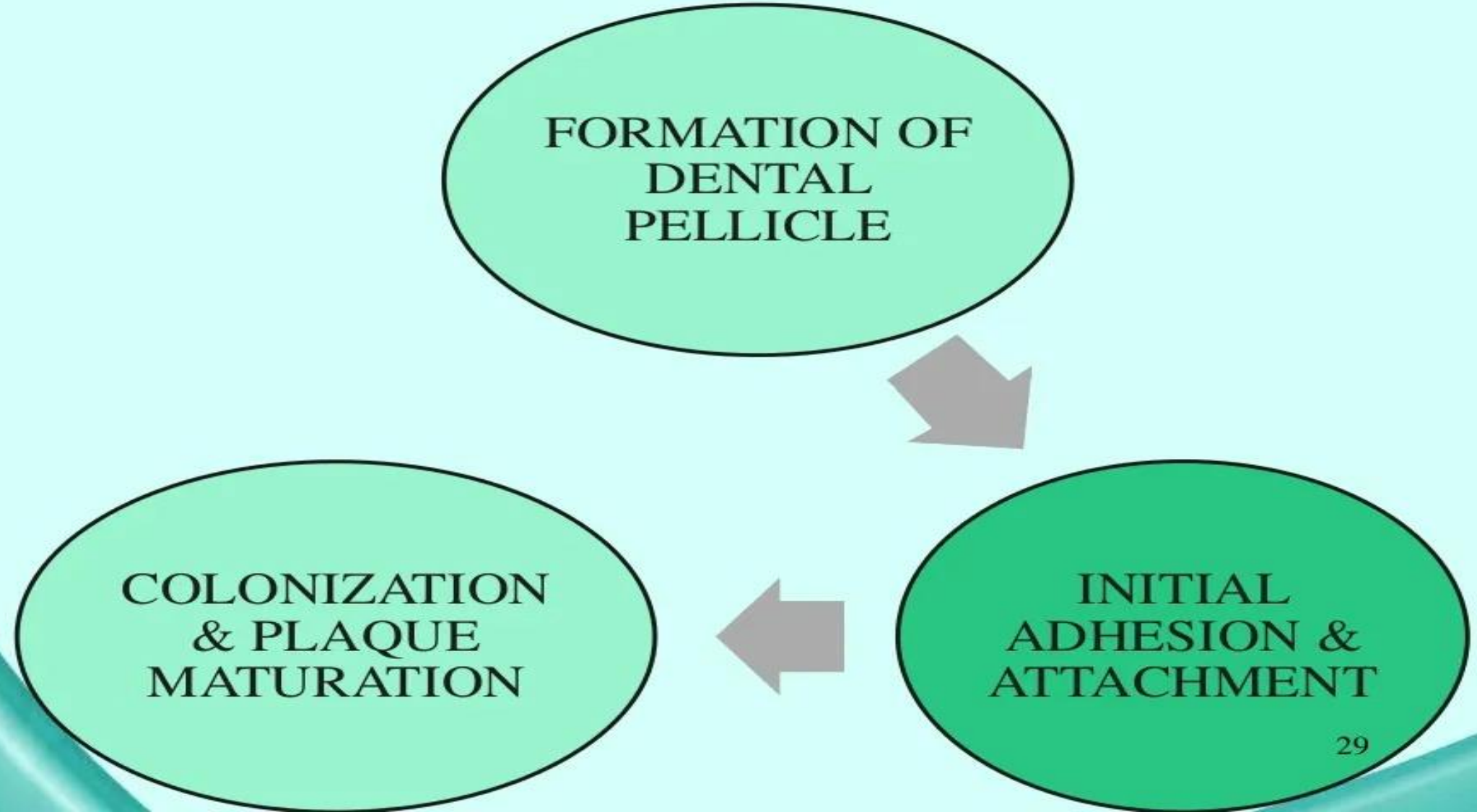
DENTA PLAQUE UNDER X 400 MAGNIFICATION



DEVELOPMENT OF DENTAL PLAQUE



Dental plaque may be readily visualized on teeth after 1-2 days of no oral hygiene.



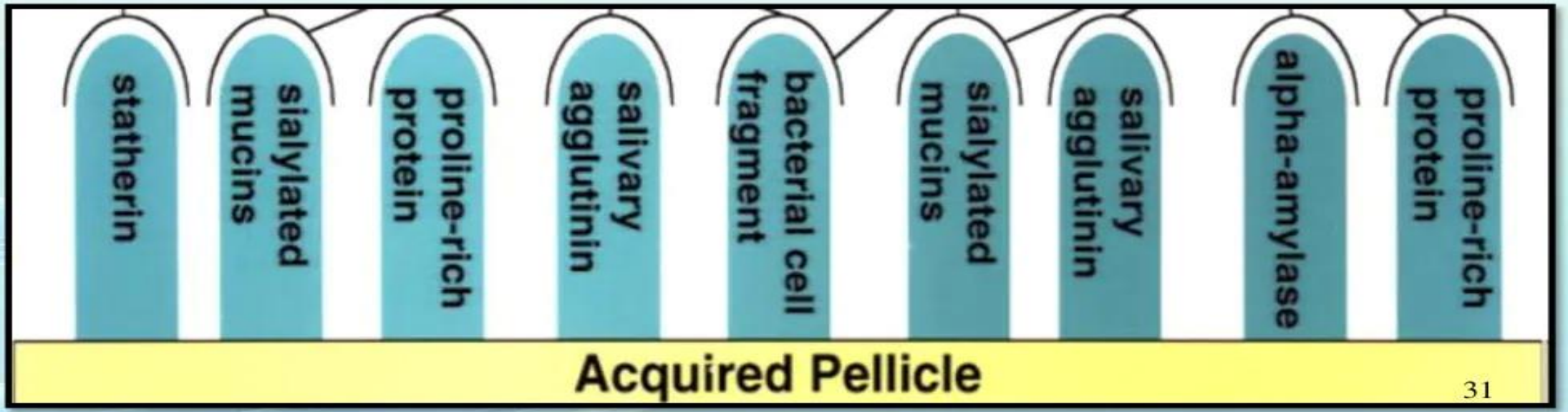
I. FORMATION OF DENTAL PELLICLE

- **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)

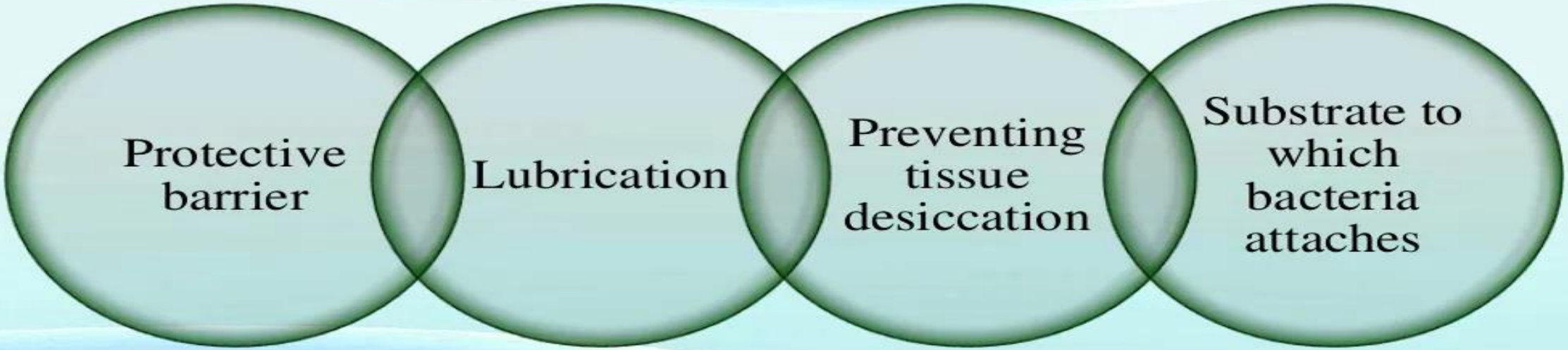
- `A fully established pellicle is found within 30 min.
  Within 24 hr, the pellicle is around 0.1-0.8 µm in diameter.

- 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.



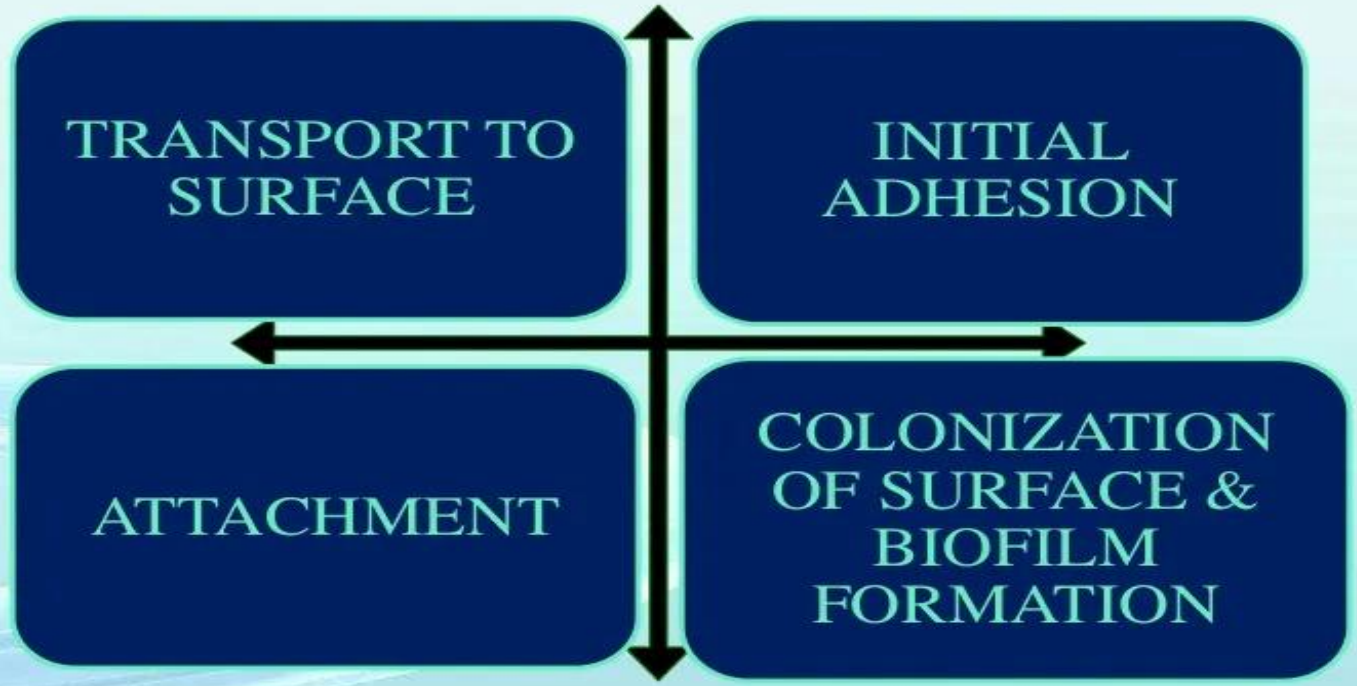
FUNCTIONS OF DENTAL PELLICLE



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
  - Brownian motion (average displacement of 40 µm/hour)
  - Sedimentation of microorganisms,
  - Liquid flow
  - 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}).**
- ($G_{TOT} = GA + GE$)

INITIAL ADHESION

- For most bacteria, G_{TOT} consists of a
  - **Secondary minimum** (where a reversible binding takes place: 5-20 nm from the surface)
  - A **positive maximum** (an energy barrier B) to adhesion
  - A **steep primary minimum** (located at <2 nm away from the surface), where an irreversible adhesion is established.

C) ATTACHMENT

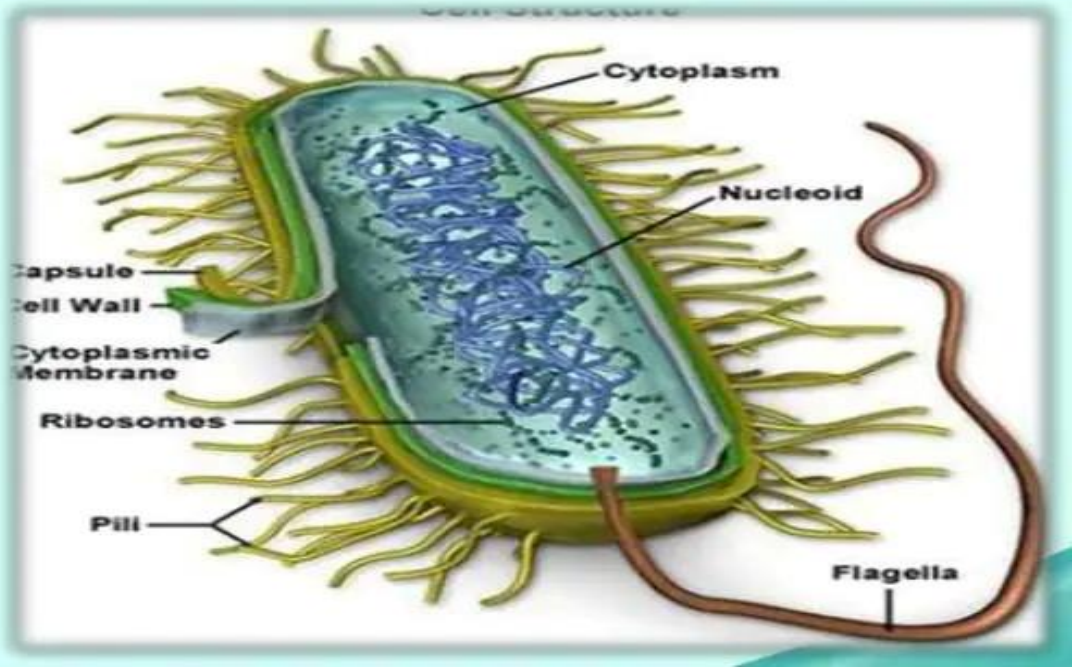
- After initial adhesion, a firm anchorage between bacterium and surface will be established by specific interactions (covalent, ionic, or hydrogen bonding).

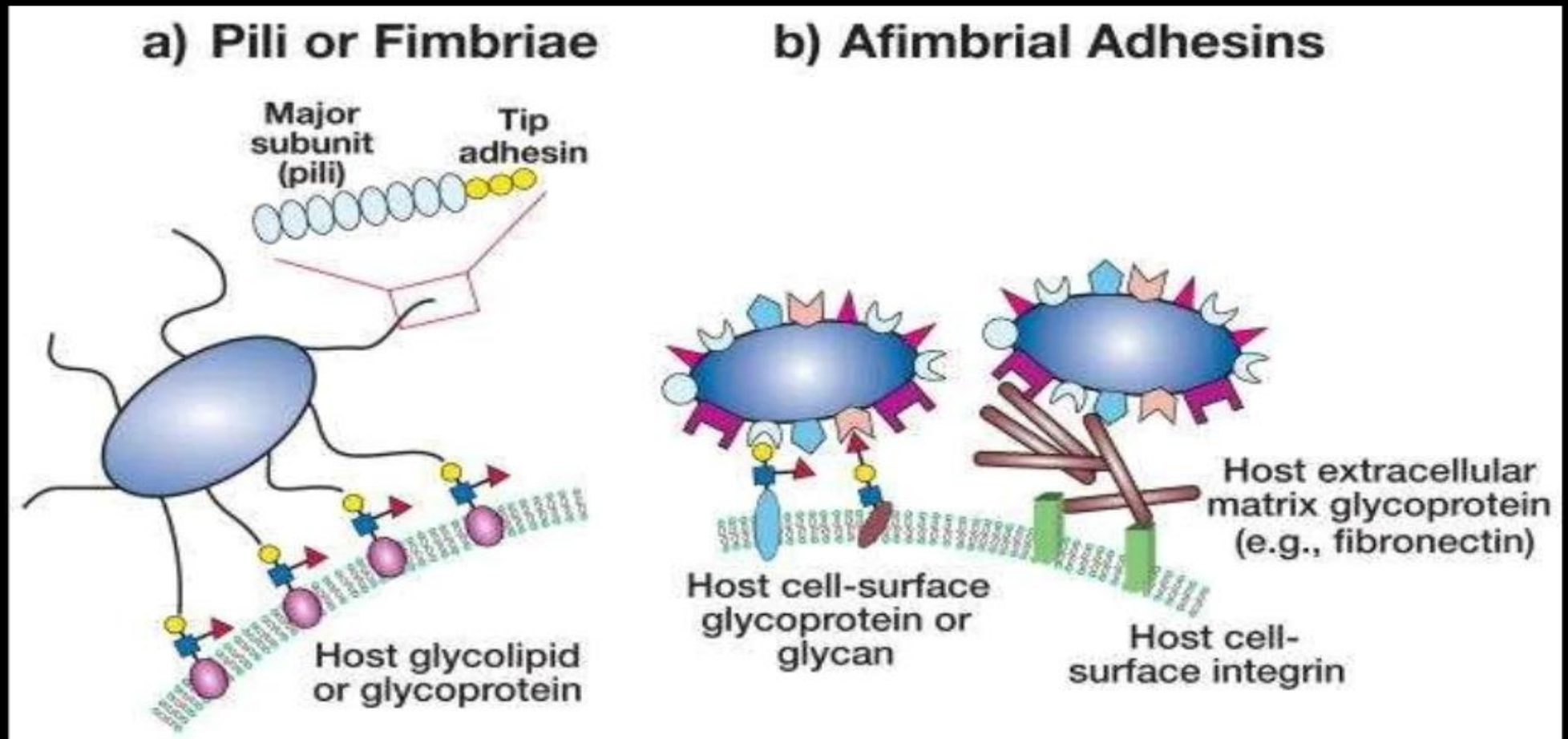
- The bonding between the bacteria & pellicle is mediated by specific extracellular components of organisms & complementary receptors on pellicle surface.

ATTACHMENT OF BACTERIA

- The attachment to tooth surface is the essential first step in the development of a biofilm.

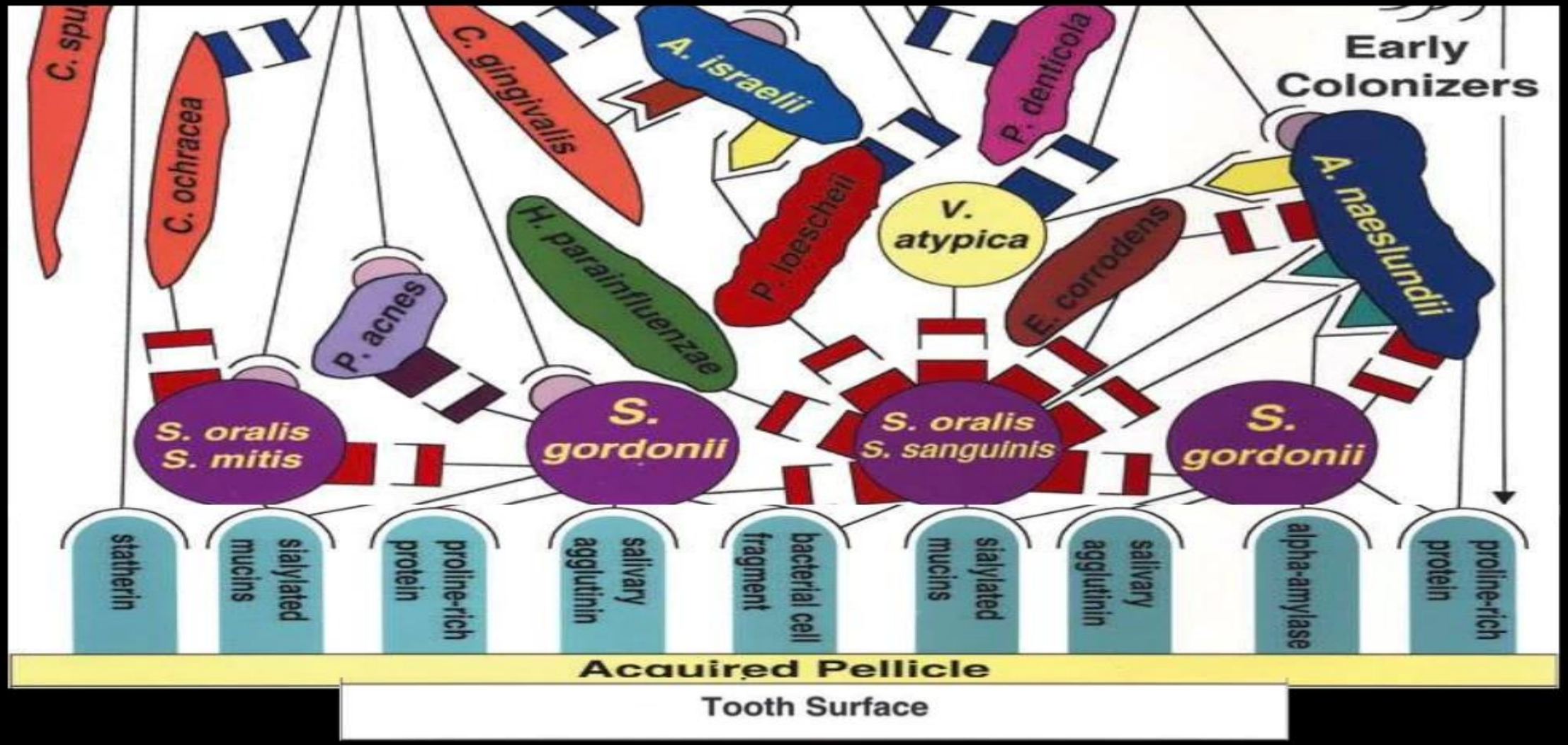
- Many bacterial species possess surface structures such as **FIMBRIAE** and **FIBRILS** that aid in their attachment to different surfaces.




a) Pili or Fimbriae
Major subunit (pili)
Tip adhesin
Host glycolipid or glycoprotein
b) Afimbrial Adhesins
Host extracellular matrix glycoprotein (e.g., fibronectin)
Host cell-surface glycoprotein or glycan
Host cell-surface integrin

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.


Early Colonizers
C. ochracea
C. gingivalis
A. israelii
P. denticola
A. naeslundii
P. loescheii
V. atypica
E. corrodens
H. parainfluenzae
P. acnes
S. oralis S. mitis
S. gordonii
S. oralis S. sanguinis
S. gordonii
statherin
sialylated mucins
proline-rich protein
salivary agglutinin
bacterial cell fragment
sialylated mucins
salivary agglutinin
alpha-amylase
proline-rich protein
Acquired Pellicle
Tooth Surface

- **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*.

COAGGREGATION

- **Coaggregation** is referred to as cell-to cell recognition of genetically distinct partner cell types.

- Occurs primarily through the **highly specific stereo chemical interaction** of protein and carbohydrate molecules located on the bacterial cell surfaces.

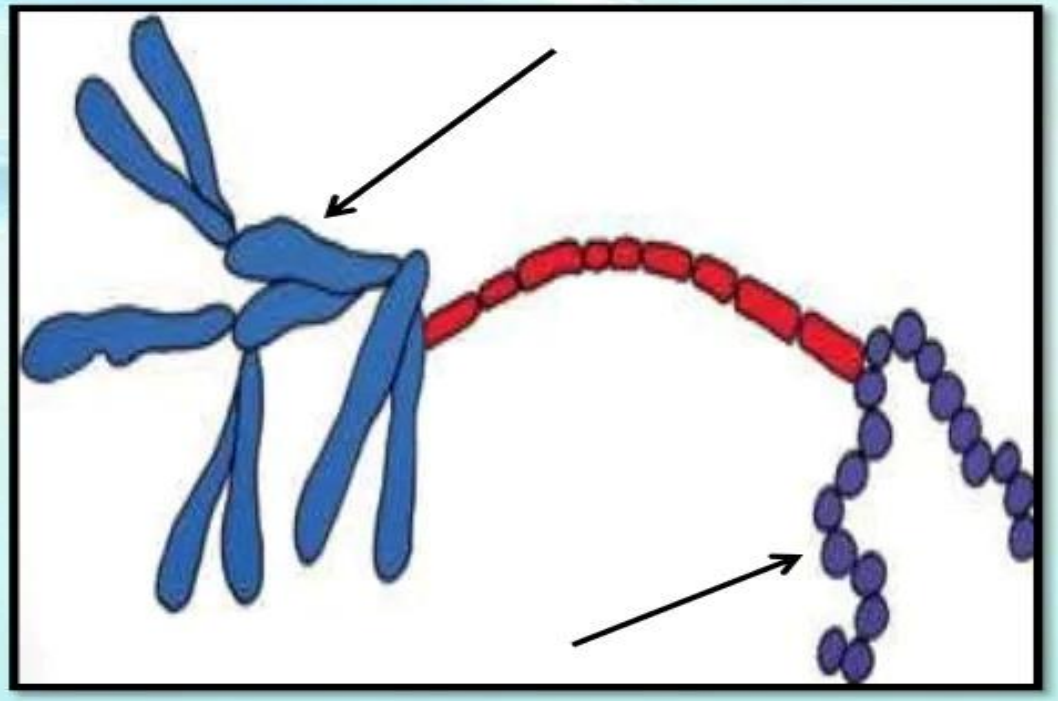
- Also by the **less specific interactions** resulting from hydrophobic, electrostatic, and van der Waals forces.

COAGGREGATION

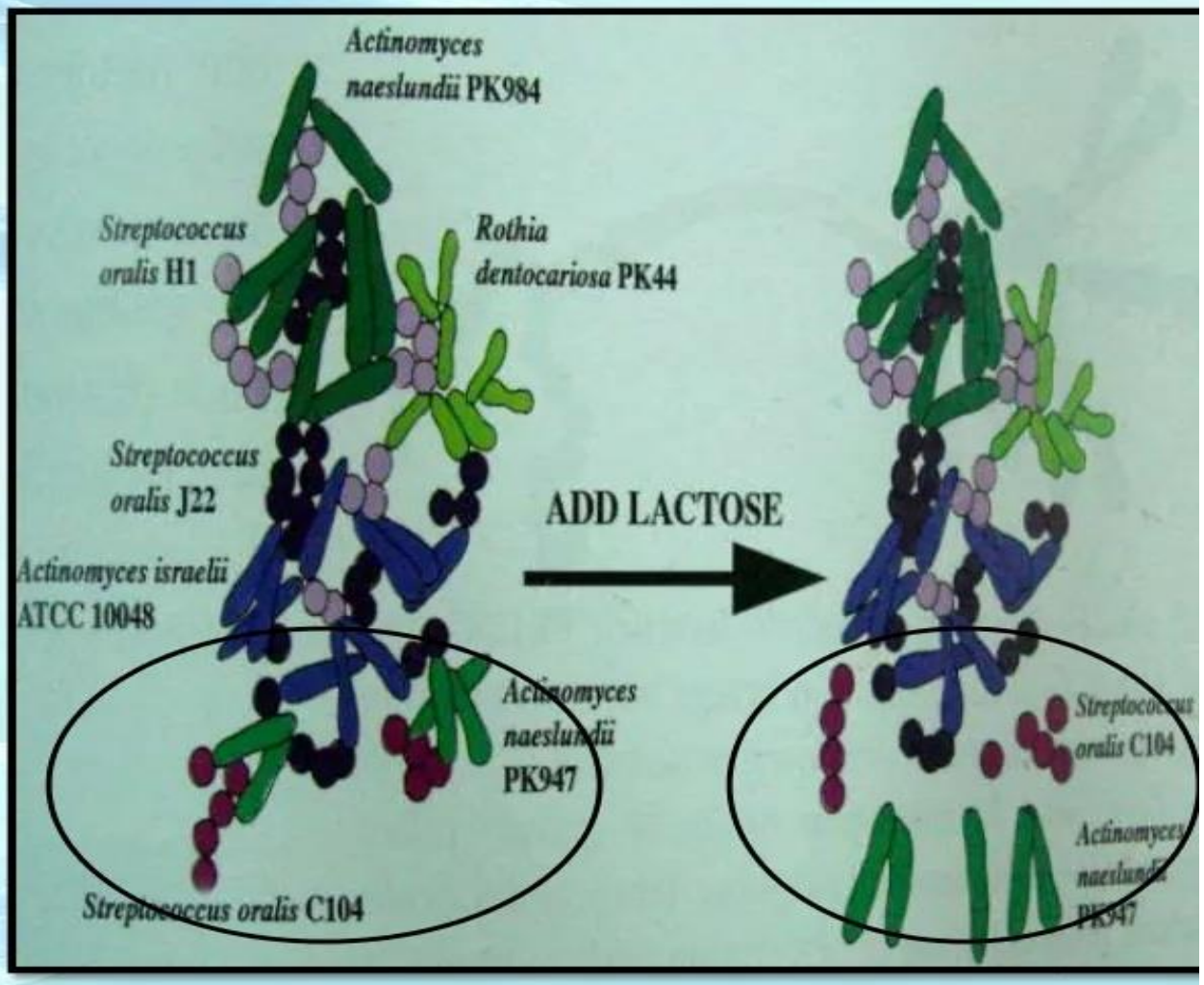
- Well characterized interaction include the coaggregation of:
  - Fusobacterium nucleatum ⟷ S. sanguis,
  - Prevotella loescheii ⟷ A. viscosus
  - Capnocytophaga ochraceus ⟷ A. viscosus
  - Streptococci show intrageneric co-aggregation → bind to the nascent monolayer of already bound streptococci.
- Later stages – coaggregation between different Gram negative species seen – F. nucleatum & P. gingivalis or T. denticola.

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



- Thus most coaggregation among strains of different genera are mediated by lectin-like adhesin & can be inhibited by lactose & other glycosides.



FACTORS THAT AFFECT SUPRAGINGIVAL DENTAL PLAQUE

Topography of Supragingival Plaque.

- Early plaque formation on teeth follows a typical topographic pattern with initial growth along the gingival margin and from the interdental spaces (i.e., the areas protected from shear forces).
- Later, a further extension in the coronal direction can be observed. This pattern may change severely when the tooth surface contains irregularities that offer a favorable growth path. Plaque formation may also originate from grooves
- The idea that coaggregation is important during the formation of oral biofilms opens new perspectives, especially for the use of probiotics.
- An analysis of more than 13,000 plaque samples that looked for 40 subgingival microorganisms using a DNA-hybridization methodology defined color-coded “complexes” of periodontal microorganisms that tend to be found together in health or disease.

Surface microroughness

- Rough intraoral surfaces (e.g., crown margins, implant abutments, denture bases) accumulate and retain more plaque and calculus in terms of thickness, area, and colony-forming units.

- plaque also reveals an increased maturity/pathogenicity of its bacterial components, which is characterized by an increased proportion of motile organisms and spirochetes and/or a denser packing of them Smoothing an intraoral surface decreases the rate of plaque formation.

- however, further smoothing does not result in an additional reduction in plaque formation.

- ***Individual Variables That Influence Plaque Formation*. The rate of plaque formation differs significantly between subjects, and these differences may overrule surface characteristics. A distinction is often made between "heavy" (fast) and "light" (slow) plaque formers**

- **a multiple regression analysis showed that the clinical wettability of the tooth surfaces, the saliva-induced aggregation of oral bacteria, and the relative salivary flow conditions around the sampled teeth explained 90% of the variation.**

- **Moreover, the saliva from light plaque formers reduced the colloidal stability of bacterial suspensions of, for example, *S. sanguinis***

- *Variation Within the Dentition.* Within a dental arch, large differences in plaque growth rate can be detected. In general, early plaque formation occurs faster: in the lower jaw (as compared with the upper jaw); in molar areas; on the buccal tooth surfaces (as compared with palatal sites, especially in the upper jaw); and
in the interdental regions

- *Impact of Gingival Inflammation and Saliva.* Several studies clearly indicate that early in vivo plaque formation is more rapid on tooth surfaces facing inflamed gingival margins than on those adjacent to healthy gingivae. These studies suggest that the increase in crevicular fluid production enhances plaque

- ***impact of Patient's Age*. Although older studies were contradictory, more recent papers clearly indicate that a subject's age does not influence de novo plaque formation.**

- **In a study by Fransson and colleagues,100 no differences could be detected in de novo However, the developed plaque in the older patient group resulted in more severe gingival inflammation, which seems to indicate an increased susceptibility to gingivitis with aging.**

- ***Spontaneous Tooth Cleaning*. Many clinicians still think that plaque is removed spontaneously from the teeth, such as during eating. However, on the basis of the firm attachment between the bacteria and the surface, this seems unlikely. Even in the occlusal surfaces of the molars, plaque remains, even after chewing**

Metabolism of Dental Plaque Bacteria

- The majority of nutrients for dental plaque bacteria originate from saliva or gingival crevicular fluid, although the host diet provides an occasional but nevertheless important food supply. The transition from gram-positive to gram-negative microorganisms observed in the structural development of dental plaque is paralleled by a physiologic transition in the developing plaque. The early colonizers (e.g., *Streptococcus* and *Actinomyces* spp.) use oxygen and lower the redox potential of the environment, which then favors the growth of anaerobic species.

- Many of the gram-positive early colonizers use sugars as an energy source. The bacteria that predominate in mature plaque are anaerobic and asaccharolytic (i.e., they do not break down sugars), and they use amino acids and small peptides as energy sources. Laboratory studies have demonstrated many metabolic interactions among the different bacteria found in dental plaque

Communication Between Biofilm Bacteria

- Bacterial cells **do not exist in isolation**. In a biofilm, bacteria have the capacity to communicate with each other. One example of this is quorum sensing, in which bacteria secrete a signaling molecule that accumulates in the local environment and triggers a response such as a change in the expression of specific genes once they reach a critical threshold concentration.
- The threshold concentration is reached only at a high cell density, and therefore bacteria sense that the population has reached a critical mass or quorum. There is some evidence that intercellular communication can occur after cell–cell contact and that, in this case, communication may not involve secreted signaling molecules..
- Two types of signaling molecules have been detected from dental plaque bacteria: peptides released by gram-positive organisms during growth and a "universal" signal molecule called *autoinducer.*

- Peptide signals are produced by oral streptococci; they are recognized by cells of the same strain that produced them and possibly also by different species of streptococci.

- In contrast with the strain-specific competence-stimulating peptides, AI-2 is produced and detected by many different bacteria.

Biofilms and Antimicrobial Resistance

- Bacteria growing in microbial communities adherent to a **surface** do not "behave" the same way as bacteria growing suspended in a liquid environment (i.e., in a planktonic or unattached state). For example, **the resistance of bacteria to antimicrobial agents is dramatically increased in the biofilm.**

- Almost without exception, **organisms in a biofilm** are 1000 to 1500 times more resistant as compared with antibiotics in their planktonic state. The mechanisms of this increased resistance differ from species to species, from antibiotic to antibiotic, and for biofilms growing in different habitats.

- It is generally accepted that the resistance of bacteria to antibiotics is affected by their **nutritional status, growth rate, temperature, pH, and prior exposure to subeffective concentrations of antimicrobial agents**. Variations in any of these parameters will lead to a varied response to antibiotics within a biofilm.