SWAMI VIVEKANAND SUBHARTI UNIVERSITY
UGC Approved Meerut
Where Education is a Passion ...
NAAC A

DENTAL PLAQUE

Dr. Mrinalini Agarwal
Senior Lecturer
MDS- Department of Periodontology
Subharti Dental College & Hospital
Swami Vivekanand Subharti Universiy
Meerut, U.P.

SWAMI VIVEKANAND SUBHARTI UNIVERSITY
UGC Approved Meerut
Where Education is a Passion ...
A NAAC

Contents:

- Definitions
- Classification
- Composition of plaque
- Microbial complexes
- Formation of plaque
- Formation of plaque in relation to time
- Theories of plaque formation
- Criteria for identification of periodontal pathogen
- Microbial shift from health to disease
- References
- Quiz time

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."

(WHO-1961)

SWAMI VIVEKANAND SUBHARTI UNIVERSITY
UGC Approved Meerut
Where Education is a Passion ...
A NAAC

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

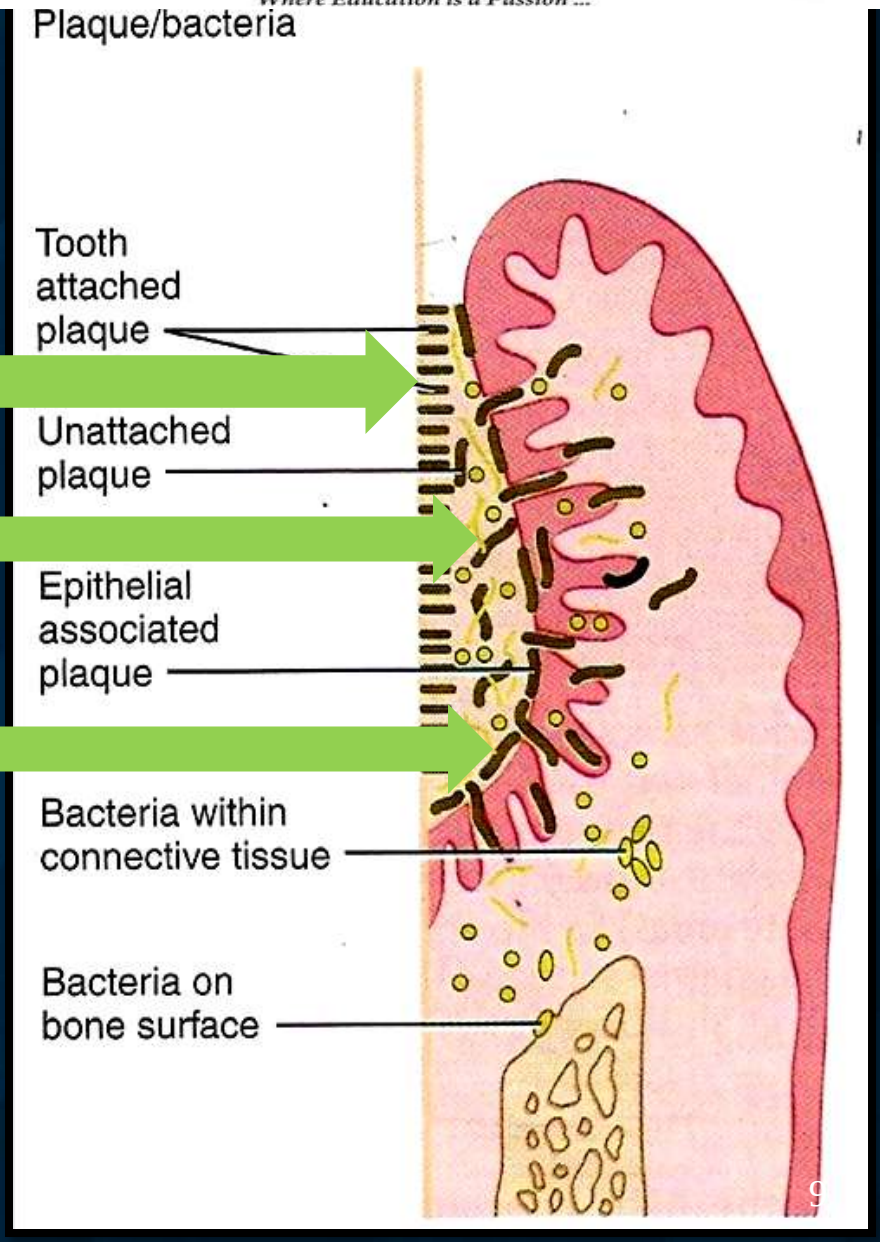
SUPRA- GINGIVAL PLAQUE

- ***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.


Plaque/bacteria
Tooth attached plaque
Unattached plaque
Epithelial associated plaque
Bacteria within connective tissue
Bacteria on bone surface

TOOTH ATTACHED	UNATTACHED	TISSUE ATTACHED
Gram positive – rods and cocci,	Gram negative rods, filaments, spirochetes	Both
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

COMPOSITION OF DENTAL PLAQUE

INTERCELLULAR MATRIX 20-30%

MICROORGANISM-80%

SWAMI VIVEKANAND SUBHARTI UNIVERSITY
UGC Approved Meerut
Where Education is a Passion ...

NAAC A

INTERCELLULAR MATRIX

ORGANIC

INORGANIC

ORGANIC MATRIX	INORGANIC MATRIX
Polysaccharide– produced by bacteria, e.g : dextran	Predominantly Ca, P- major Na, K, F - trace
Protein -albumin	source of inorganic material in supra-gingival plaque is primarily saliva.
Glycoprotein-from saliva	source of inorganic material in sub gingival plaque is GCF
Lipid	

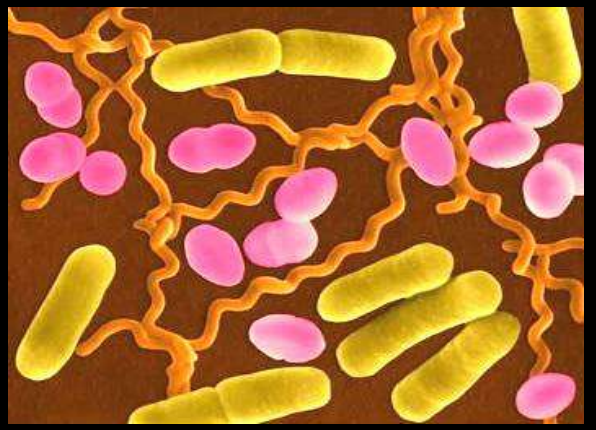
MICROORGANISMS

- One gram of plaque contains approximately 2×10^{11} bacteria.

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

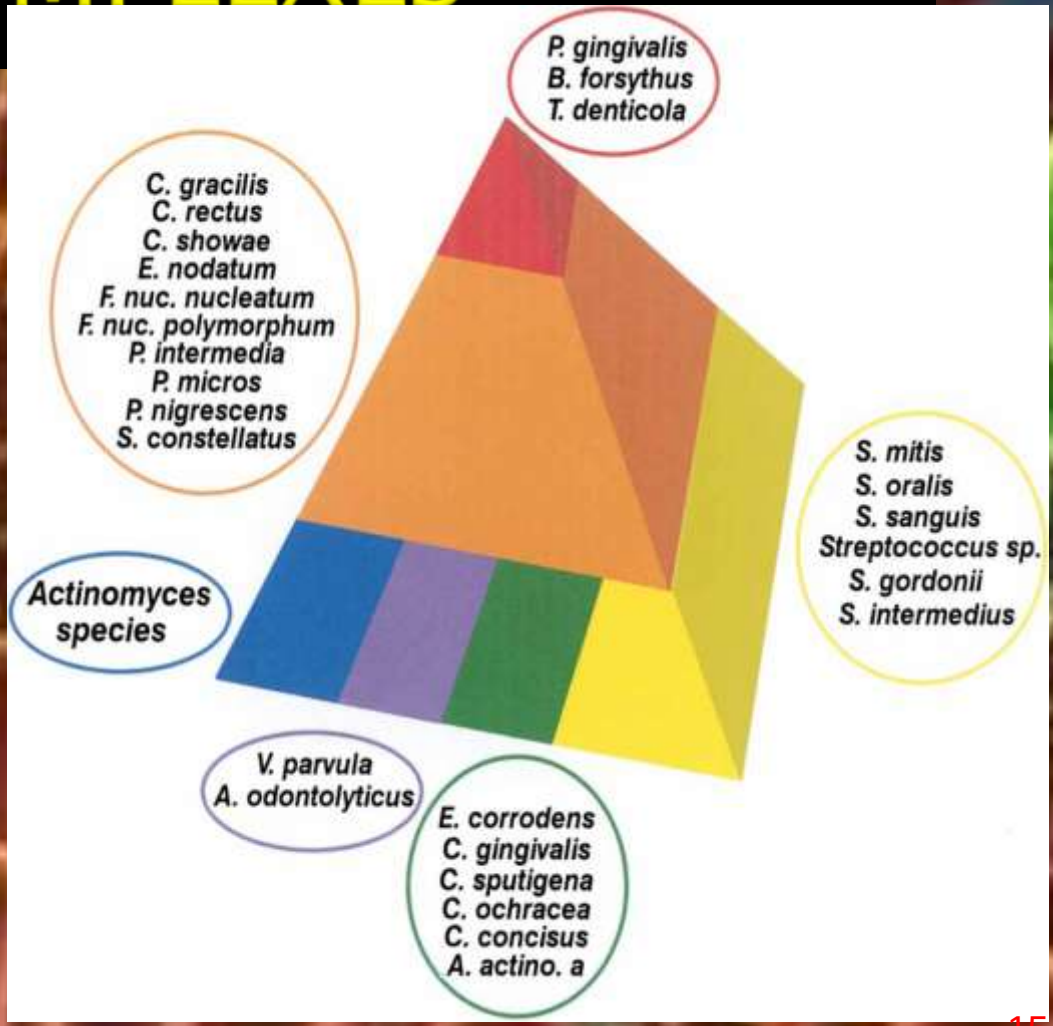
- *More than 500 distinct microbial species found in dental plaque- Moore 1994*

Nonbacterial organisms are:
MYCOPLASMA
YEAST
PROTOZOA
VIRUSES



MICROBIAL COMPLEXES

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

16

SECONDARY COLONIZERS

C.rectus

P.Intermedia
P.Nigrescens
P.Micros
F.nucleatum

E.nodatum

C.showae

E.Corrodens
Capnocyptophaga spp
A.actinomycetemcomitans

P.Gingivalis
B.Forsythus
T.denticola

17

SILVER COMPLEX

- HSV type 1
- EBSTEIN BARR VIRUS
- HUMAN CYTOMEGALO VIRUS

lar Resistance

Bacterial cell

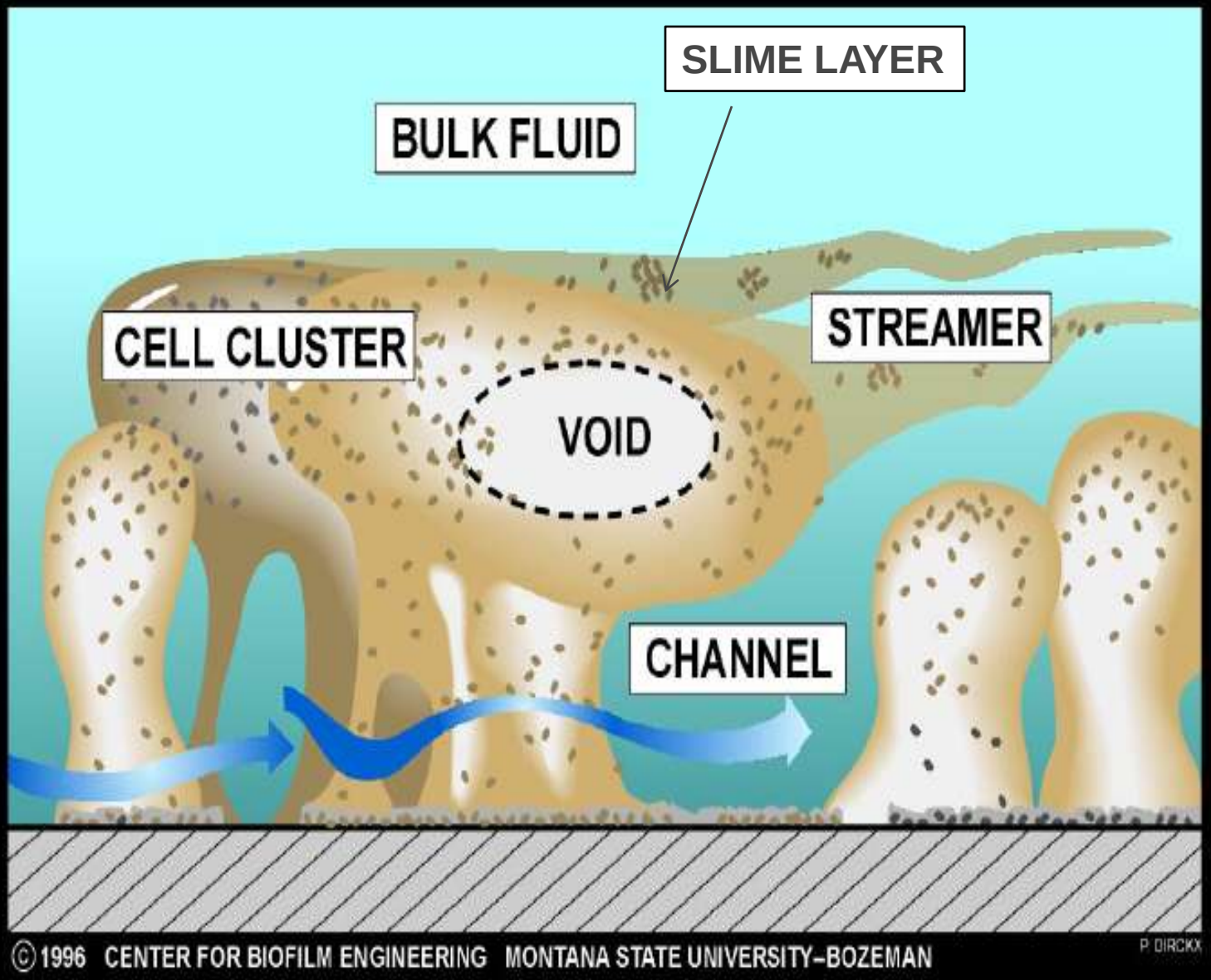
Cell Membrane

Biofilms

" 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.


SLIME LAYER
BULK FLUID
CELL CLUSTER
STREAMER
VOID
CHANNEL
© 1996 CENTER FOR BIOFILM ENGINEERING MONTANA STATE UNIVERSITY-BOZEMAN
P. DIRCKX

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

- Quorum sensing in bacteria , ‘‘involves the regulation of expression of specific genes through the accumulation of signaling compounds that mediate intercellular communication.”

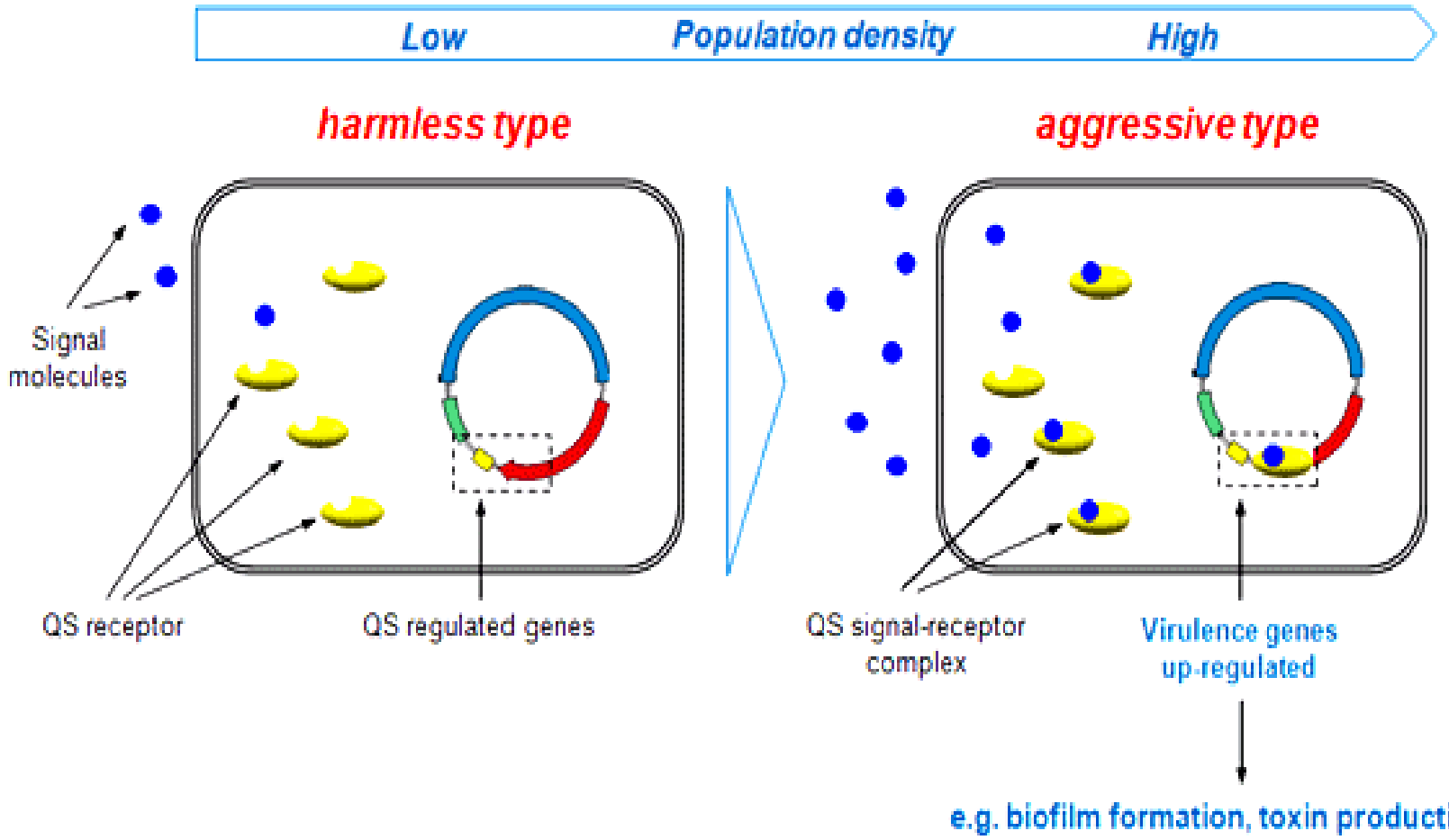
  (Prosser 1999)

- 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



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….!!!!


Breathe
IN
OUT

SWAMI VIVEKANAND SUBHARTI UNIVERSITY
UGC Approved Meerut
Where Education is a Passion ...
NAAC A

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

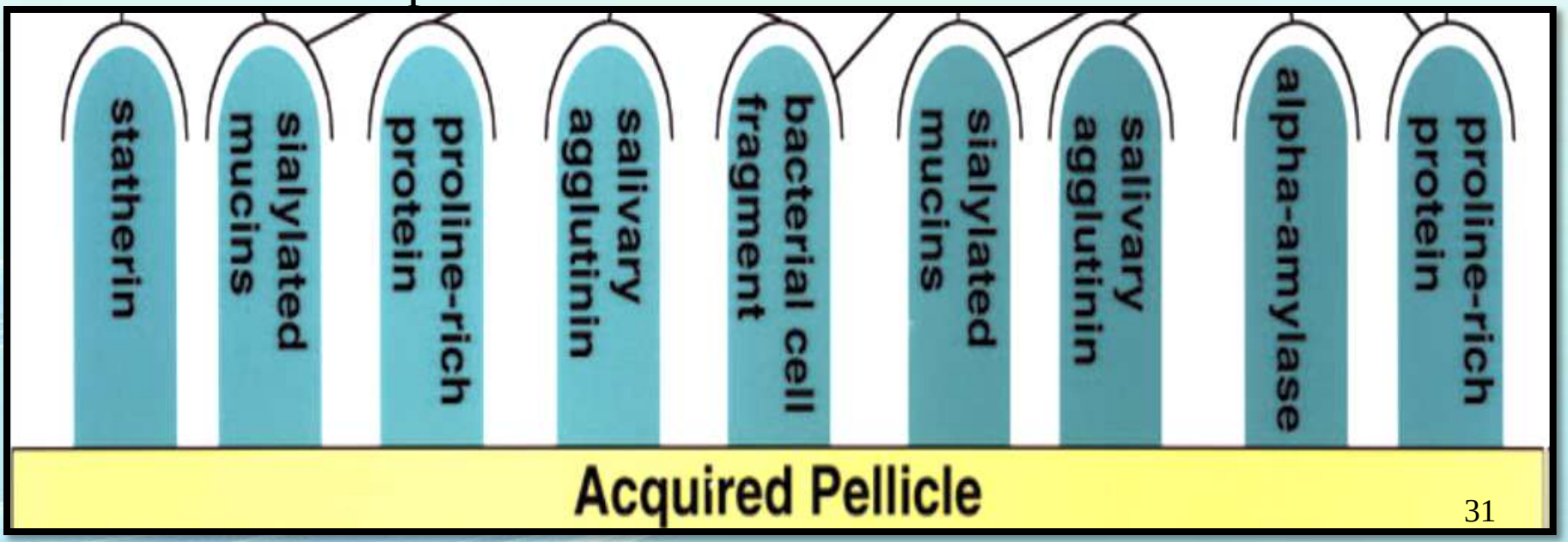
- **Acquired pellicle** may be defined as a homogenous, membranous, acellular film that covers the tooth surface and frequently form the interface between the tooth ,the dental plaque and calculus . (SCHLUGER)

- `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.



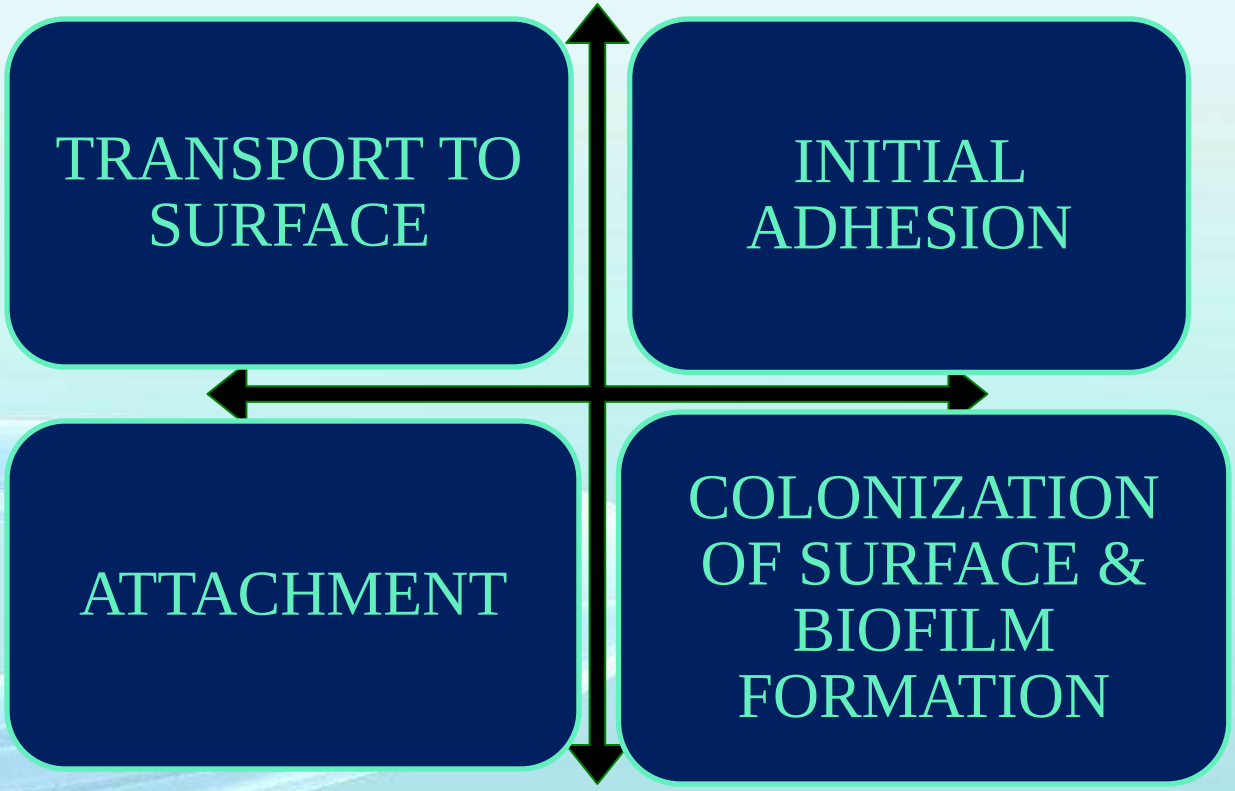
FUNCTIONS OF DENTAL PELLICLE

- 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
  - 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)

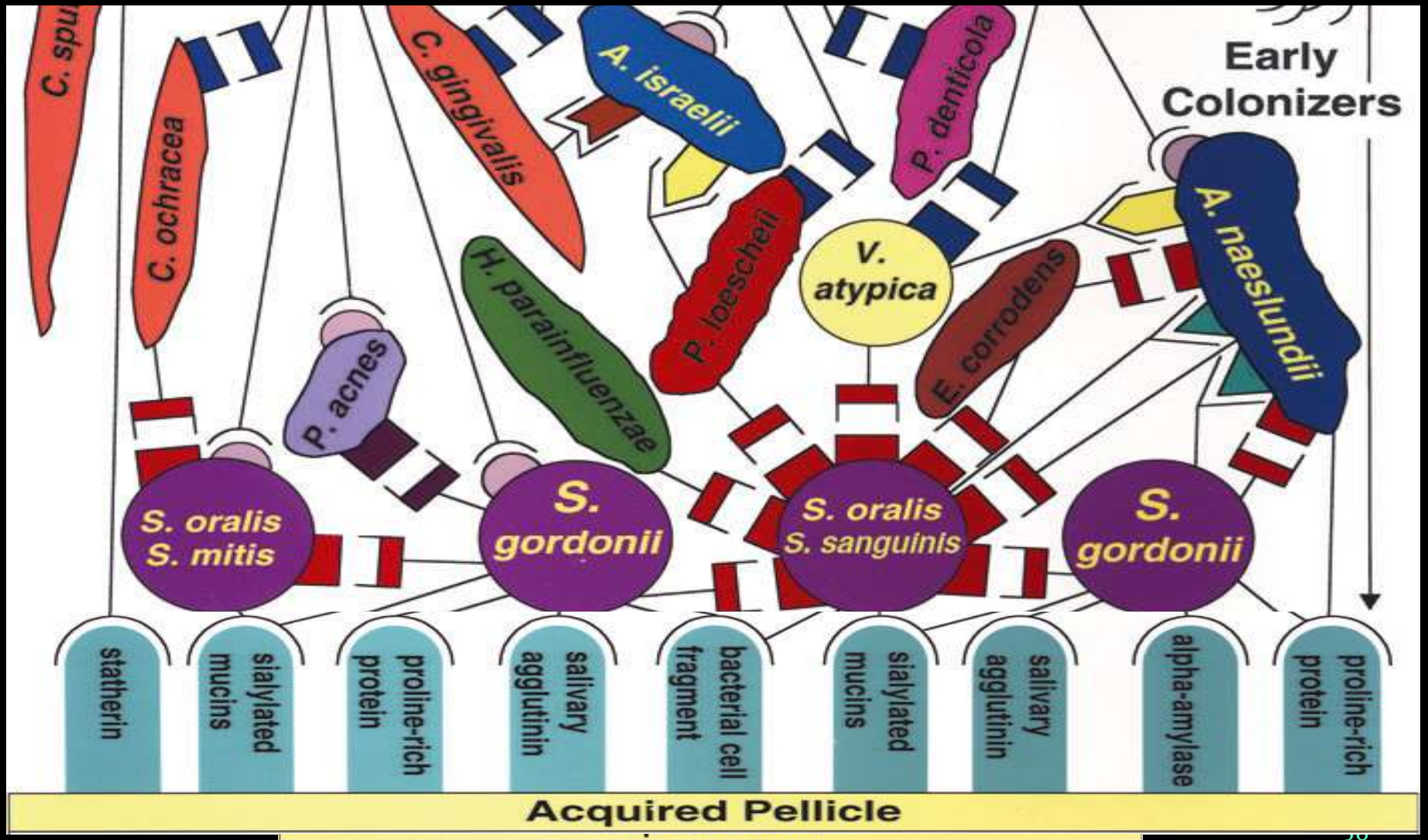
C) ATTACHMENT

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

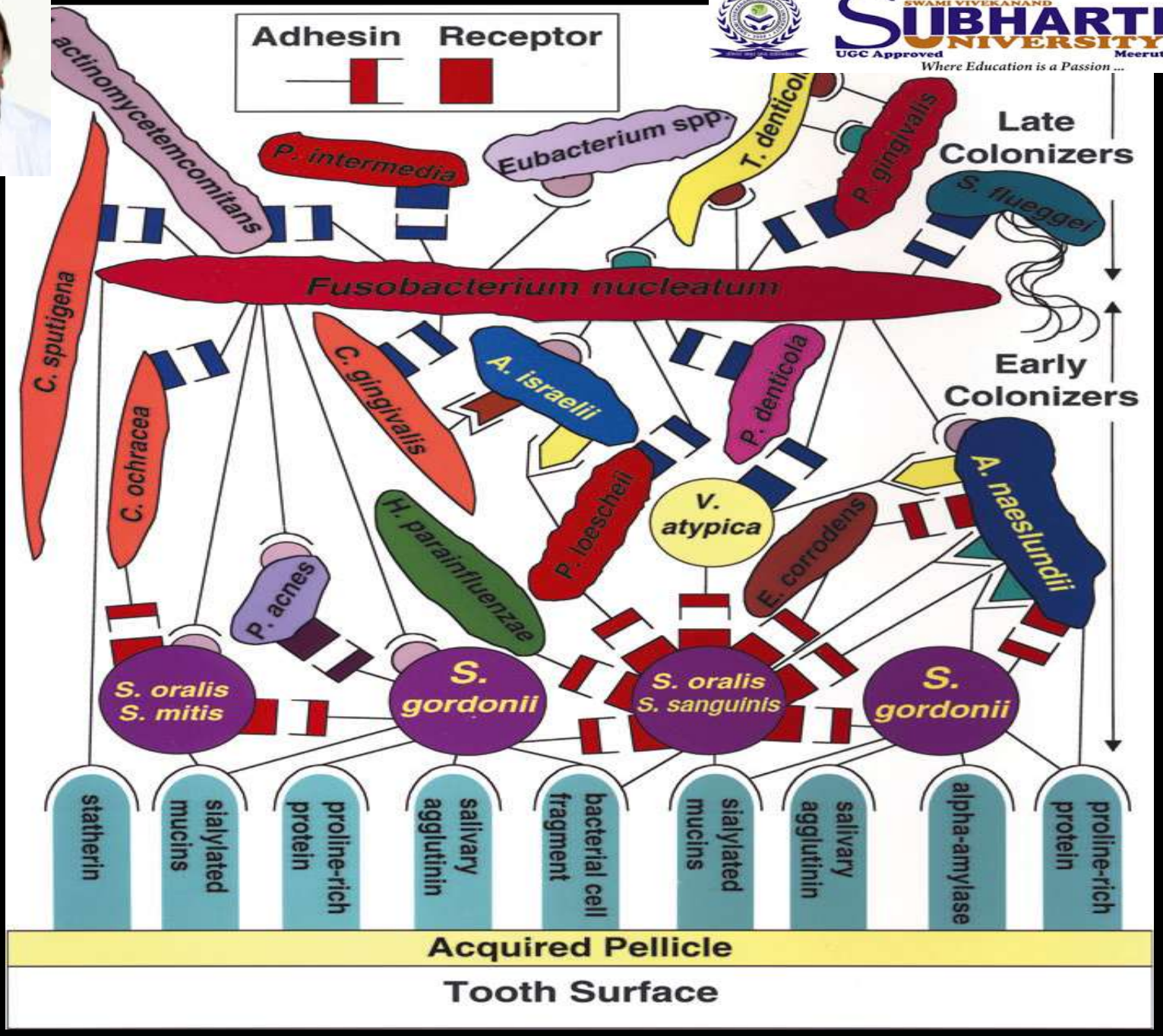
- The bonding between the bacteria & pellicle is mediated by specific extracellular components of organisms & complementary receptors on pellicle surface.

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.


Early Colonizers
C. ochracea
C. gingivalis
A. israelii
P. denticola
P. loescheii
V. atypica
E. corrodens
A. naeslundii
P. acnes
H. parainfluenzae
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

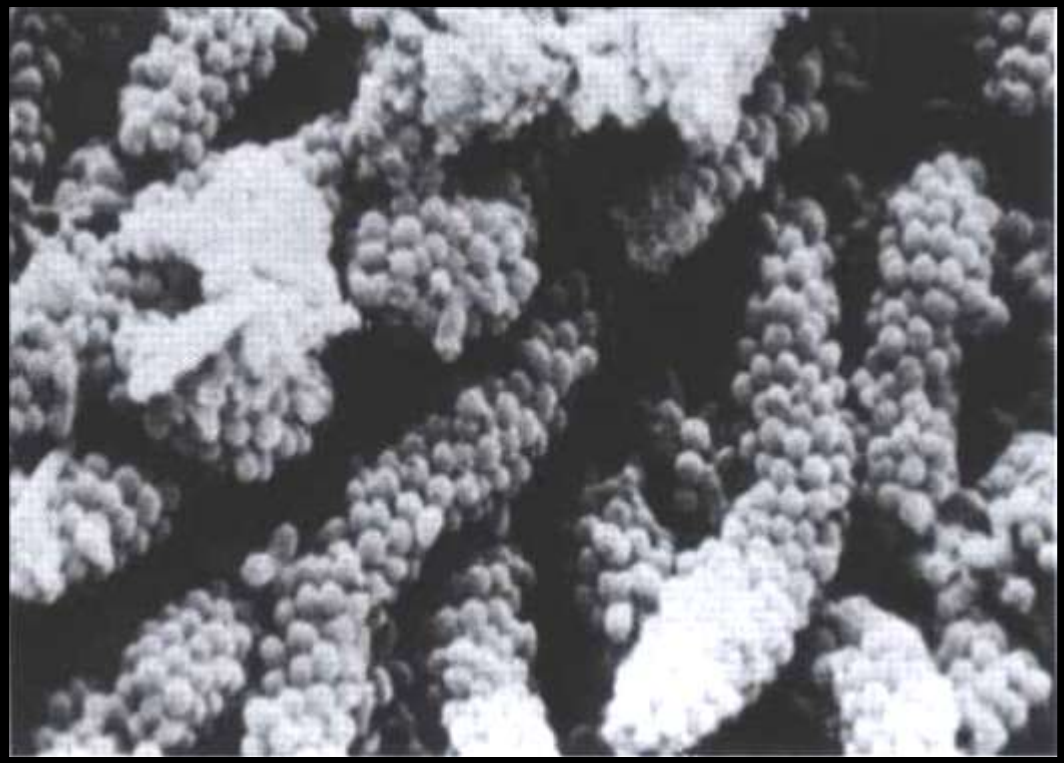
- **Secondary colonizers** are the microorganisms that do not initially colonize clean tooth surfaces, including *Prevotella intermedia*, *Prevotella loescheii*, *Capnocytophaga spp*., *Fusobacterium nucleatum*, and *Porphyromonas gingivalis*.


Adhesin
Receptor
actinomycetemcomitans
P. intermedia
Eubacterium spp.
T. denticola
P. gingivalis
S. fluegge
Late Colonizers
Fusobacterium nucleatum
C. sputigena
C. ochracea
C. gingivalis
A. israelii
P. denticola
P. loescheii
V. atypica
H. parainfluenzae
P. acnes
E. corrodens
A. naeslundii
Early Colonizers
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

GGREGATION

- 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.



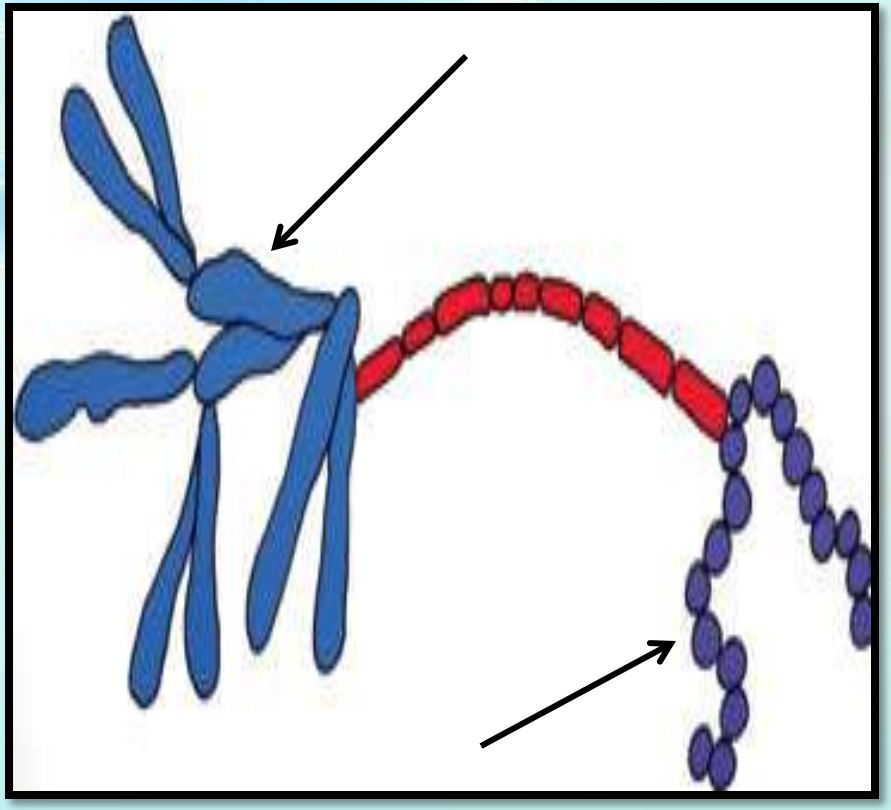
CORNCOB STRUCTURE

TEST TUBE BRUSH

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

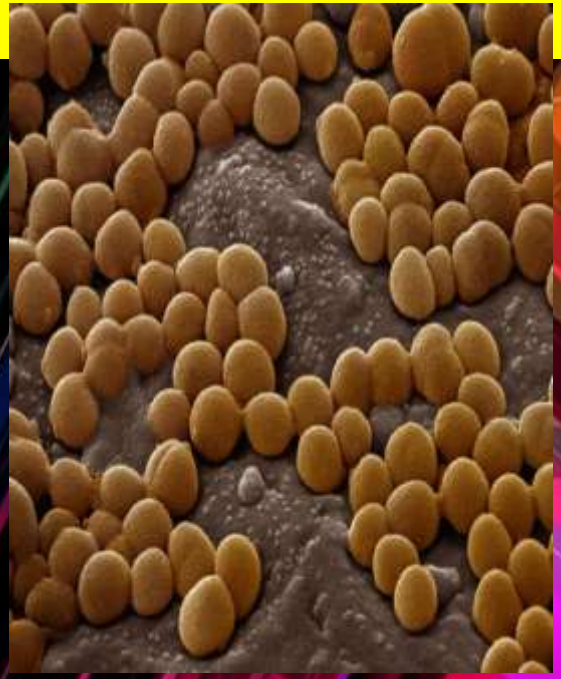


SWAMI VIVEKANAND SUBHARTI UNIVERSITY
UGC Approved Meerut
Where Education is a Passion ...
NAAC A

DENTAL PLAQUE FORMATION – RELATION TO TIME

1 HOUR

- Bacteria adhere to pellicle, and pellicle coats the enamel.
- ***Gram positive rods*** and ***cocci*** are laid down in the first hour.



24-48 HOUR

- Bacteria multiply and form mini-colonies in layers upon the pellicle.
- The bacteria adhere and increase in mass and thickness.

4-7 DAYS

- 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.

7-14 DAYS

- 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.

14-21 DAYS

- ***Vibrio and spirochetes*** continue to multiply.
- The bacteria become
  - ✓Highly organized
  - ✓Filamentous
  - ✓Perpendicular to the tooth surface
- The signs of inflamed gums are obvious

SWAMI VIVEKANAND SUBHARTI UNIVERSITY
UGC Approved Meerut
Where Education is a Passion ...
A NAAC

DETECTION OF PLAQUE

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

- 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.

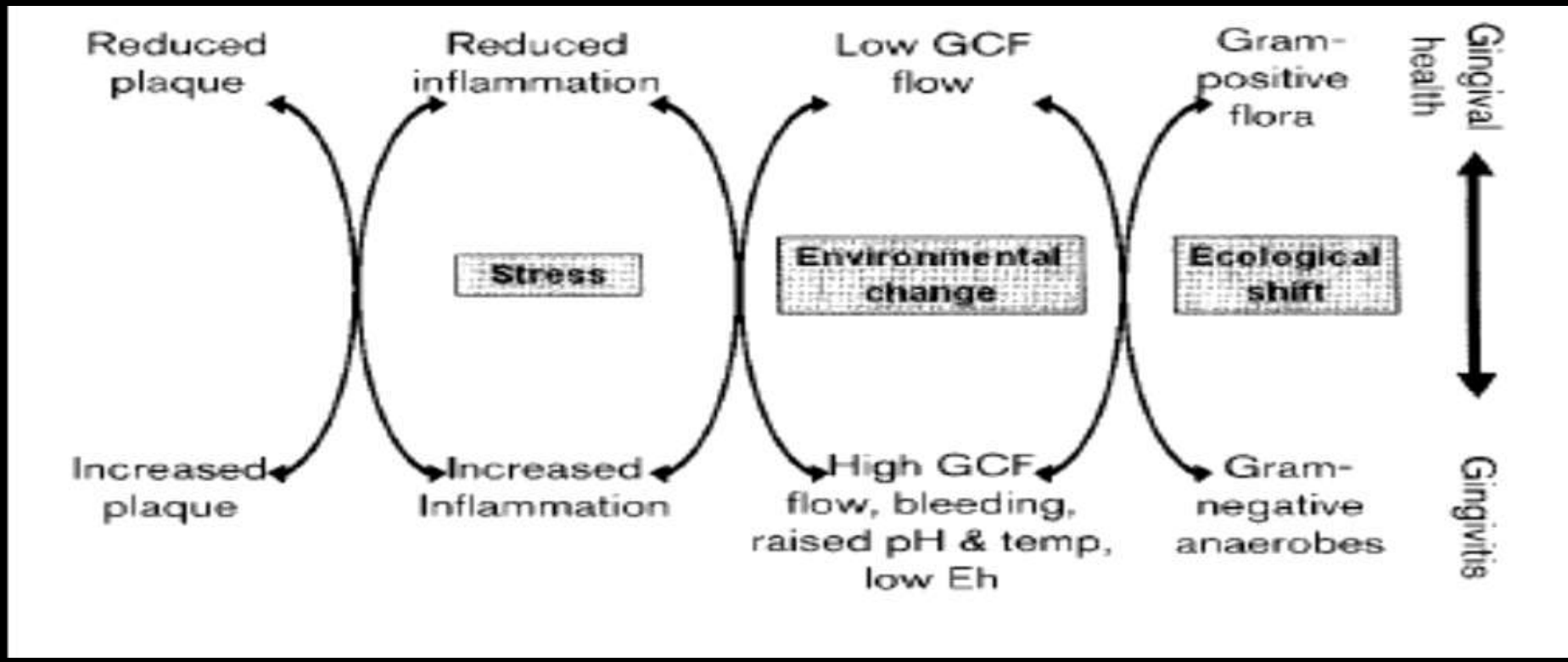
- Plaque harboring specific bacterial pathogens results in periodontal disease.
- ***A. actinomycetemcomitans*** is a pathogen in aggressive periodontitis.

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
Gingival health
Stress
Environmental change
Ecological shift
Increased plaque
Increased Inflammation
High GCF flow, bleeding, raised pH & temp, low Eh
Gram-negative anaerobes
Gingivitis

CRITERIA FOR IDENTIFICATION OF PERIODONTAL PATHOGENS

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

SWAMI VIVEKANAND SUBHARTI UNIVERSITY
UGC Approved Meerut
Where Education is a Passion ...
ACCREDITED WITH GRADE A NAAC

REFERENCES

- Clinical Periodontology - Carranza (9th & 10th edn)
- Clinical Periodontology- Jan Lindhe, Thorklid Karring , Niklaus P Lang
- Clinical Periodontology : Listgarten
- Periodontal microbial ecology - Perio 2000,Vol. 38, 2005, 135–187

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

- Dental biofilms: difficult therapeutic targets - Periodontology 2000, Vol. 28, 2002, 12–55

- Dental biofilms: difficult therapeutic targets - Periodontology 2000, Vol. 28, 2002, 12–55

DENTAL PLAQUE MCQs

1. Defintion 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

SWAMI VIVEKANAND SUBHARTI UNIVERSITY
UGC Approved Meerut
Where Education is a Passion ...
NAAC A

THANK YOU

FOR LISTENING

Dr. Mrinalini Agarwal
Email id: mrinaliniagarwal1@gmail.com