Khadija. K. Al-Dulaimy **Microbiology /College Of Dentistry**
B.Sc., M.Sc., Med. Microbiol. **Al- Anbar University**

Dental plaque and Dental caries.

Dental plaque is a general term for the complex microbial community that develops on the tooth surface, embedded in a matrix of polymers of bacterial and salivary origin. Plaque is composed of organic, inorganic materials derived from saliva, gingival crevicular fluid & bacterial products. The organic constituents of plaque include polysaccharides, proteins, glycoproteins & lipid material. The inorganic constituents of plaque include primarily of calcium & phosphorus & traces of sodium, potassium. Plaque that becomes calcified is referred to as calculus or tartar. The majority of plaque is found associated with the protected and stagnant regions of the tooth surface such as fissures, approximal regions between teeth, and the gingival crevice .

Plaque causes cavities when the acids from plaque attack teeth after eating. With repeated acid attacks, the tooth enamel can break down and a cavity may form. Plaque that is not removed can also irritate the gums around the teeth, leading to gingivitis (red, swollen, bleeding gums), periodontal disease and tooth loss.

- Specific plaque hypothesis states that, not all plaque is pathogenic and its pathogenicity depends on the presence of certain specific microbial pathogens in the plaque.

Types of Dental Plaque:

Dental plaque is broadly classified based on its position as:
a) Supragingival plaque: is found at or above the gingival margin
b) Sub gingival plaque: is found below the gingival margin.

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

a) Formation of Dental Pellicle
b) Initial colonization of bacteria
c) Secondary colonization & plaque maturation.

What is Dental Pellicle?

Pellicle is a glycoprotein, derived from components of saliva and crevicular fluid as well as from bacteria and host tissue cell products and debris. Pellicle is formed on all surfaces of the oral cavity, including all tissue surfaces as well as surfaces of teeth and fixed and removable restorations if any. Pellicle functions as a protective barrier but pellicle provides a substrate on which bacteria progressively accumulate to form Dental Plaque. Pellicle provides a medium or base on which bacteria in the oral cavity attach. Pellicle gets easily stained & may display many colors ranging from white to dark brown due which the teeth appear discolored.

Mechanism of Pellicle formation:

The enamel surface has predominance of negatively charged phosphate groups that interact directly or indirectly with positively charged components of salivary & crevicular fluid macromolecules. Within few hours bacteria are found on the pellicle. These initial bacteria colonizing the pellicle-coated tooth surface are predominantly gram-positive microorganisms such as Actinomyces viscosus & Streptococcus sanguis. These initial colonizers adhere to the pellicle through specific molecules termed adhesins, on the bacterial surface that interact with receptors in dental pellicle. The major constituents of pellicle are salivary glycoproteins, phosphoproteins and lipids, including statherin, amylase, proline-rich peptides (PRPs) .For example, cells of Actinomyces viscosus possess fibrous protein structures called Fimbriae that extend from the bacterial cell surface. Protein adhesins on these Fimbriae specifically bind to proline-rich proteins that are found in dental pellicle, resulting in the attachment of the bacterial cell to the pellicle-coated tooth surface.

The plaque mass then matures through the growth of attached species as well as colonization & growth of additional species. Microorganisms are generally transported passively to the tooth surface by the flow of saliva; few oral bacterial species are motile (e.g. possess flagella), and these are mainly located subgingivally. Plaque may be readily visualized on teeth after 1 to 2 days with no oral hygiene measures. Plaque is white, grayish or yellow & has a globular appearance. With good oral hygiene practices one can remove this pellicle & plaque layers. There are different types of treatments which are extensively aim at removal of plaque &stains; patient friendly methods are practically more acceptable than clinical treatment .Certain chemical toothpastes remove stains by altering the surface environment of the teeth thereby inhibiting the adherence of plaque. For example hydrogen peroxide in the toothpaste acts by releasing nascent oxygen, abrasive characteristic of baking soda or silica is said to cleanse the tooth surface. Some toothpaste contains specific

enzymes which are believed to remove plaque, stain & thereby improving the gingival health. These toothpastes are biologically compatible with the tooth surface & the surrounding soft tissues.

the initial colonizers constitute a highly selected part of the oral microflora. Within minutes, coccal bacteria appear on the surface , and these pioneer organisms are mainly streptococci, especially members of the mitis-group of streptococci(e.g. *S. sanguinis, S. oralis* and *S. mitis* biovar) . *Streptococcus sanguinis* and *S. oralis* produce an IgA1 protease which may help them to survive and overcome a key element of the host defences during the early stages of plaque formation. *Actinomyces* spp. are also commonly isolated after 2 hours, as are *Haemophilus* spp. and *Neisseria* spp., while obligately anaerobic species are detected only rarely at this stage and are usually in low numbers. Some aggregates of mixtures of cells may also attach .Once attached, these pioneer populations start to divide and form microcolonies; these early colonizers become embedded in bacterial extracellular slimes and polysaccharides together with additional layers of adsorbed salivary proteins and glycoproteins. The early streptoccocal colonizers (mitis-group of streptococci) possess a range of glycosidase activities that enable them to interact and use salivary glycoproteins as substrates . The fastest rates of multiplication occur during these early stages of plaque formation.

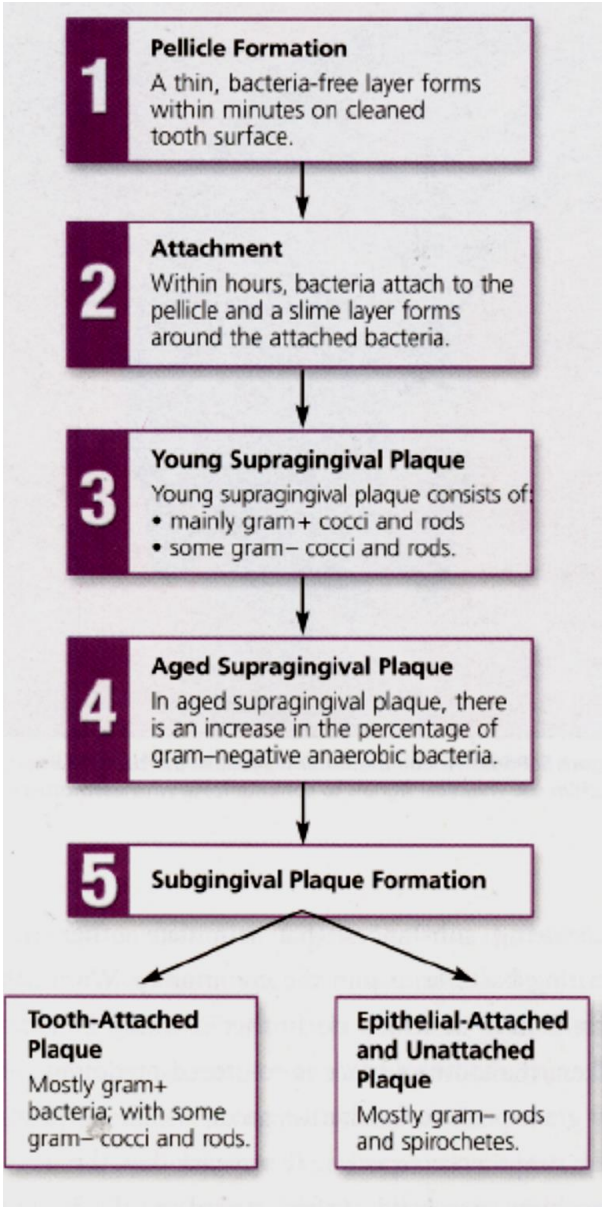
Table 5.2 Proportions of bacteria in developing supragingival plaque.

Bacterium	Time of plaque development (h)		
	2	24	48
Streptococcus sanguinis	8	12	29
Streptococcus oralis	20	21	12
mutans streptococci	3	2	4
Streptococcus salivarius	<1	<1	<1
Actinomyces naeslundii	6	7	5
Actinomyces odontolyticus	2	3	6
Haemophilus spp.	11	18	21
Capnocytophaga spp.	<1	<1	<1
Fusobacterium spp.	<1	<1	<1
Black-pigmented anaerobes	0	<0.01	<0.01

The initial bacteria that colonize the pellicle surface are mostly;

- gram+ve
- Facultative microorganism such as actinomyces viscosus and streptococcus sanguis.

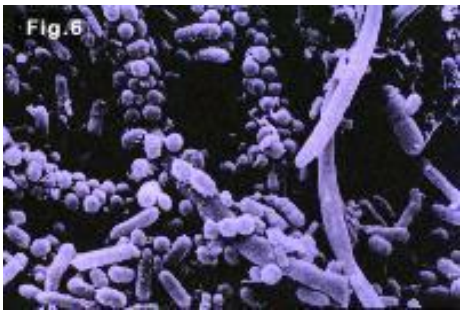
The adhesion of organisms to the 'conditioned' tooth surface is a complex process involving, initially, weak electrostatic attractive forces, followed by a variety of specific molecular interactions between bacterial adhesins and receptors adsorbed to the surface (acquired pellicle). These latter processes together with the synthesis of extracellular polysaccharides (EPS) from, for example, sucrose serve to increase the probability of permanent attachment. EPS also contributes to the plaque matrix. Pioneer species interact directly with the acquired pellicle while subsequent biofilm formation is dependent on intra and intergeneric coadhesion between bacteria (involving lectin-mediated binding) and the subsequent growth of the attached microorganisms. If conditions become unfavourable, some cells are able to actively detach, providing the opportunity to colonize other sites.



The pattern of plaque biofilm development can be divided into three phases:

1. Attachment of bacteria to a solid surface;
2. Formation of microcolonies on the surface; and
3. Formation of the mature, subgingival plaque biofilms.

The bi adhere to the previously attached cells. The bacteria cluster together to form sessile,mushroom-shaped micro colonies that are attached to the tooth surface at a narrow base.



In particular, specific associations of different bacterial forms have been observed. For example, the adherence of cocci to filaments results in a typical form referred to as "test-tube brushes" or "corn-cob" arrays and these structures can be seen in Figure 6.The result of coaggregation is the formation of a complex array of different bacteria linked to one another. Following a few days of undisturbed plaque formation, the gingival margin becomes inflamed and swollen. These inflammatory changes result in the creation of a deepened gingival sulcus. The biofilm extends into this subgingival region and flourishes in this protected environment, resulting in the formation of a mature subgingival plaque biofilm. Gingival inflammation does not appear until the biofilm changes from one composed largely of gram-positive bacteria to one containing gram-negative species such as *Fusobacterium nucleatum*, *Prevotella intermedia*, and *Capnocytophaga* species. A subgingival bacterial microcolony, predominantly composed of gram-negative anaerobic bacteria, becomes established in the gingival sulcus between 3 and 12 weeks after the beginning of supragingival plaque formation. Most bacterial species currently suspected of being periodontal pathogens are anaerobic, gram-negative bacteria.

Synergistic interactions

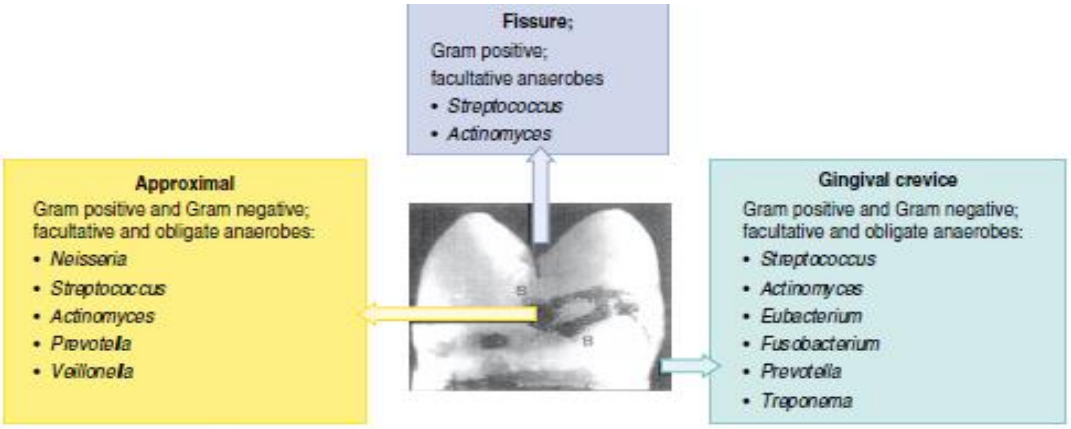
The structural interactions of the bacteria probably are a reflection of the complex metabolic interactions that are known to occur between different plaque microorganisms. One example of this is the production of succinic acid from *Campylobacter* species that is known to be used as a growth factor by *Porphyromonas gingivalis*. *Streptococcus* and *Actinomyces* species produce formate, which may then be used by *Campylobacter* species. *Fusobacterium* species produce both thiamine and isobutyrate that may be used by spirochetes to support their growth. The metabolic and structural interactions between different plaque microorganisms are a reflection of the incredible complexity of this ecological niche.

Antagonistic interactions

Antagonism is a major contributing factor in determining the composition of microbial ecosystems such as dental plaque . The production of antagonistic compounds (such as bacteriocins or bacteriocin-like substances, BLIS) can give an organism a competitive advantage when interacting with other microbes. Bacteriocins are relatively high molecular weight proteins that can inhibit the growth of related bacteria

while the producer strains are resistant to the action of the bacteriocins they produce. Bacteriocins are produced by most species of oral streptococci (e.g. mutacin by *S. mutans* and sanguicin by *S. sanguinis*), as well as by *C. matruchotii*, black-pigmented anaerobes, and *A. actinomycetemcomitans*; in contrast, *Actinomyces* species are not generally bacteriocinogenic. Although bacteriocins are usually limited in their spectrum of activity, many of the streptococcal bacteriocins are broad spectrum, inhibiting species belonging to Gram positive(including *Actinomyces*) and Gram negative genera. The production of bacteriocins may give strains a competitive advantage during colonization.Other inhibitory factors produced by plaque bacteria include organic acids, hydrogen peroxide, and enzymes. The production of hydrogen peroxide by members of the mitis-group of streptococci has been proposed as a mechanism whereby the numbers of periodontal pathogens are reduced in plaque to levels at which they are incapable of initiating disease. Some periodontal pathogens (e.g. *A. actinomycetemcomitans*) produce factors inhibitory to oral streptococci, might result from an ecological imbalance between dynamically-interacting groups of bacteria. The low pH generated from carbohydrate metabolism is also inhibitory to many plaque species, particularly Gram negative organisms and to some streptococci associated with sound enamel.

Specific microbial species that are important in plaque development and disease development are outlined below based on their categorization by cell wall morphology (Gram-positive, Gram-negative, or spirochetal) and their physiological status (facultative or anaerobic).



Predominant groups of bacteria found at distinct sites on the tooth surface.

The major differences between supragingival and subgingival

SUPRAGINGIVAL PLAQUE.	SUBGINGIVAL PLAQUE.
Contains 50% matrix	Has little or no matrix
It contains mostly gram+ve	Mostly gram-ve
Has few motile bacterial	Motile bacterial is common
It's aerobic unless it's thick	Highly anaerobic area is present
It metabolizes predominantly carbohydrates.	Predominantly metabolizes protein.

EFFECTS ON THE PERIODONTIUM

- Supragingival plague if allowed to grow can lead to gingivitis and it influences the growth, accumulation and pathologic potential of subgingival plaque.
- Subgingival plaque is associated with enlarged gingiva, calculus formation, periodontitis etc.

CARIOGENICITY OF PLAQUE

- The presence of cariogenic bacteria
- The ability of the plaque to adhere to the tooth surface.
- Its ability to resist dissolution by saliva
- Its protection of bacterial acids from salivary buffering.

Examination.

You can rely on your dentist to tell you if you have dental plaque, but you can also take the do-it-yourself route. Disclosing tablets, swabs and solution are plaque-revealing products that can be used at home. These products temporarily stain dental plaque so that you can see where it is and how much there is on your teeth.

How It Is Done

You will brush and floss your teeth. Then you will use the disclosing product. Follow the instructions on the package. Gently rinse your mouth with water. Check your teeth for plaque that has been colored with the dye. Your gums also may be stained and appear red, but this is not a problem. Use a dental mirror, if you have one, to help see behind teeth and the areas in the back of your mouth. If you find stained plaque, brush and floss again until it is gone. This helps you find areas you are missing.

Disclosing tablets

Chew a disclosing tablet and allow it to mix with your saliva. Swish the mixture around in your mouth with your tongue for about 30 seconds and then spit it out.

Disclosing solution

Put some disclosing solution in your mouth, swish it around for about 30 seconds, and then spit the solution out.

Disclosing swabs

Apply the swab to all tooth surfaces in your mouth. Use these products regularly until you find no more areas of stained plaque after you brush and floss. You may want to test for plaque once a month to be sure you are getting rid of the plaque.

Prevention

Follow these tips on how to remove plaque from teeth :

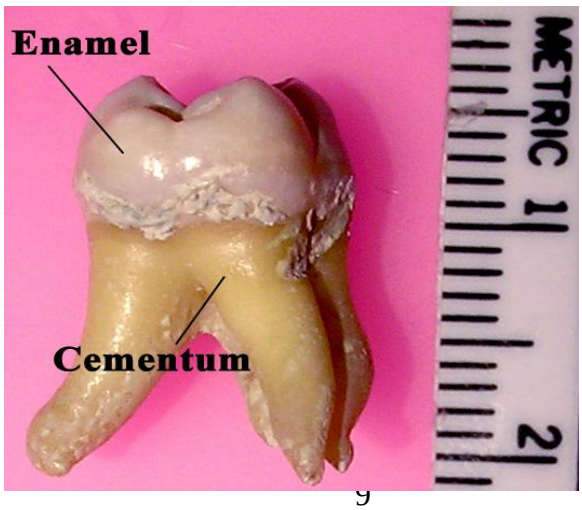
- Brush thoroughly at least twice a day, with a fluoride toothpaste, to remove plaque from your teeth
- Use dental floss daily to remove plaque from between your teeth and under your gum line, where your toothbrush may not reach
- Check your teeth with plaque disclosing tablets to ensure removing tooth plaque.
- Control your diet. Limit sugary or starchy foods, especially sticky snacks
- Ask your dentist or dental hygienist if your plaque removal techniques are ok.
- Visit your dentist regularly for professional cleanings and dental examinations
- Ask your dentist if a dental sealant is appropriate for you. Dental sealants are a thin, plastic coating that are painted on the chewing surfaces of teeth to protect them from cavities and decay.
- Use of an antibacterial mouth rinse can reduce bacteria that cause plaque and gum disease, according to the American Dental Association.

Due to the structure of biofilms, physical removal of bacterial plaque biofilms is the most effective means of control. Subgingival plaque within pockets cannot be reached by brushes, floss, or oral rinses. Therefore, frequent periodontal debridement of subgingival root surfaces by a dental hygienist or dentist is an essential component in the treatment of periodontitis.

Calculus

Calculus, or tartar, is the term used to describe calcified dental plaque. It consists of intra- and extracellular deposits of mineral, including apatite, brushite, and whitlockite, as well as protein and carbohydrate. Mineral growth can occur around any bacteria; areas of mineral growth can then coalesce to form calculus which may become covered byan unmineralized layer of bacteria. Calculus can occur both supragingivally (especially near the salivary ducts) and subgingivally, where it may act as an additional retentive area for plaque accumulation, thereby increasing the likelihood of gingivitis and other forms of periodontal disease. Calculus can be porous leading to the retention of bacterial antigens and the stimulation of bone resorption by toxins from periodontal pathogens. Over 80% of adults have calculus, and its prevalence increases with age. An elevated calcium ion concentration in saliva may predispose some individuals to be high calculus formers. Once formed, huge removal forces are required to detach calculus; this removal takes up a disproportionate amount of clinical time during routine visits by patients to the dentist. Consequently, a number of dental products are now formulated to restrict calculus formation. These products contain pyrophosphates, zinc salts, or polyphosphonates to inhibit mineralization by slowing crystal growth and reducing coalescence.

The inorganic content of plaque is greatly increased with the development of calculus. The process of calculus formation involves the calcification of dental plaque. The practical consequences of calculus formation are that the deposit is significantly more difficult to remove once calcified, and it leaves a rough surface on the root which is easily colonized by plaque. The calculus on the root surface of an extracted tooth is seen in Figure 2. Note the brown to black coloration of the subgingival calculus that extends to the apex of the distobuccal root, in contrast to the whitish color of the supragingival calculus.



Major Differences

Supragingival	Subgingival
• Above the gingiva	• Below the gingiva
• White, yellow	• Brown/ greenish black
• From salivary secretion	• From gingival exudates
• Salivary protein is present	• Salivary protein is absent
• Sodium content is less with the depth of pocket.	• Sodium content increases with the depth of pocket.

TREATMENT

Scaling and polishing

DENTAL CARIES

Dental caries can be defined as the localized destruction of the tissues of the tooth by bacterial fermentation of dietary carbohydrates. Cavities begin as small demineralized areas below the surface of the enamel ; once enamel has been affected, caries can progress through the dentine and into the pulp . Demineralization of the enamel is caused by acids, particularly lactic acid, produced from the microbial fermentation of dietary carbohydrates. Lesion formation involves dissolution of the enamel and the transport of the calcium and phosphate ions away into the surrounding environment. The initial stages of caries are reversible and remineralization can occur, particularly in the presence of fluoride. oral bacteria converted dietary carbohydrates into acid which solubilized the calcium phosphate of the enamel to produce a caries lesion.

The bacteria most responsible for dental cavities are the mutans streptococci, most prominently *Streptococcus mutans* and *Streptococcus sobrinus*, and lactobacilli. If left untreated, the disease can lead to pain, tooth loss and infection.[1] Today, caries remain one of the most common diseases throughout the world. Cariology is the study of dental caries.

What causes dental caries?

- Diet: Intake of fermentable carbohydrates, especially sucrose.
- Host: Quantity and quality of saliva, the quality of tooth, etc.
- Microflora: Infection by a specific set of cariogenic bacteria within dental plaque.

Mutans Streptococci

- The most prominent species found in the oral cavity include S. mutans, S. sanguis, S. mitoir, S. salivarius, and S. milleri.
- MS bacteria comprise the most important group of streptococci implicated in caries etiology.

Ecology of Mutans Streptococci

- MS does not colonize the mouth of infants prior to the eruption of teeth.
- Likewise it disappears from the mouth following extraction of all teeth.
- Infants most likely become infected from their parents or from other individuals with whom they have frequent contact since the **organisms** are not found free-living in nature and it has only been isolated from humans and certain animals.
- Infants whose mother's harbor higher levels of MS in saliva become colonized more readily than infants of mothers with low salivary S. mutans levels.
- MS are transmitted vertically, from mother to child.
- Mother's with high caries experience tend to have children with high caries.
- The genetic inheritance from both the mother and father has an influence on caries susceptibility of the child.
- MS does not colonize teeth uniformly.
- The organism may be more frequently isolated from fissures and interproximal surfaces, those areas most frequently involved in caries, than from buccal or lingual smooth surfaces.
- In addition, some smooth surfaces may consistently harbor detectable concentrations of the organisms where as comparable surfaces on other teeth in the same mouth do not.
- This suggest that MS does not spread readily from one tooth surface to another.

Virulence determinants of the mutans streptococci

- Acidogenicity and aciduricity:

- *Acidogenicity* is the ability of bacteria to produce acid from fermentable carbohydrates.

- *Aciduricity* is the ability of bacteria to grow and metabolize under highly acidic conditions.

How cariogenic bacteria cause caries?

- Acid production (Acidogenicity) Lower the pH to below 5.5, the critical pH, which drives the dissolution of calcium phosphate (hydroxyapatite) of the tooth enamel.
- Inhibit the growth of beneficial bacteria.
- Further lower the pH, promote progression of the carious lesion.
- Acid tolerance (aciduricity).
- Allows the cariogenic bacteria to thrive under acidic conditions while other beneficial bacteria are inhibited.
- This results in dominance of the plaque by cariogenic bacteria.
- The capacity of oral bacteria to tolerate acidic environment is of a major importance to the ecology of plaque communication and is directly related to caries pathogenesis**.**

SM and response to acid

- SM has constitutive acid tolerance properties:

-The aciduricity is highly attributable to an enzyme called F-ATPase.

-Extrude protons allowing the organism to maintain an adequate pH when the external pH decreases to 4.0 and lower.

Sucrose metabolism of S. mutans

- The most important substrate for the involvement of S. mutans in the caries process is the disaccharide sucrose.
- Sucrose not only serves as a primary energy source but it is also permit the initiation of additional biochemical events which are responsible for the cariogenic potential of this organism.

Lactobacillus

- Lactobacillus are strong acid producers and are among the most aciduric and acidogenic bacteria. .
- Lactobacillus may not be involved in caries initiation but rather may become secondary invaders, which contribute to the progression of already existing lesions**.**
- This was supported by the observation that lactobacilli are not detectable in plaque covering incipient lesions in smooth tooth surfaces.

Filamentous bacteria

- Several types of filamentous organisms will initiate root caries in experimental animals.
- Actinomyces and rothia species have been found in dental plaque and root caries.
- Actinomyces viscosus is an acidogenic bacterial and is often among the predominant flora of plaque covering root surfaces.

Gram negative cocci

- Veillonella: this organism has anti carious activity because it utilizes the lactic acid and converts it to propionic and other weak acids.

Tooth decay disease is caused by specific types of bacteria that produce acid in the presence of fermentable carbohydrates such as sucrose, fructose, and glucose. The mineral content of teeth is sensitive to increases in acidity from the production of lactic acid. To be specific, a tooth (which is primarily mineral in content) is in a constant state of back-and-forth demineralization and remineralization between the tooth and surrounding saliva. For people with little saliva, especially due to radiation therapies that may destroy the salivary glands, there also exists remineralization gel. These patients are particularly susceptible to dental caries. When the pH at the surface of the tooth drops below 5.5, demineralization proceeds faster than remineralization (meaning that there is a net loss of mineral structure on the tooth's surface). Most foods are in this acidic range and without remineralization, this results in the ensuing decay. Depending on the extent of tooth destruction, various treatments can be used to restore teeth to proper form, function, and aesthetics, but there is no known method to regenerate large amounts of tooth structure. Instead, dental health organizations advocate preventive and prophylactic measures, such as regular oral hygiene and dietary modifications, to avoid dental caries.

S. mutans, S. sanguis, and S. mitis are, besides *Actinomyces viscosus* and *A. naeslundii,* responsible for dental caries . These streptococci can attach to the proteins covering the tooth enamel, where they then convert sucrose into extracellular polysaccharides (mutan, dextran, levan). These sticky substances, in which the original bacterial layer along with

secondary bacterial colonizers are embedded, form dental plaque. The final metabolites of the numerous plaque bacteria are organic acids that breach the enamel, allowing the different caries bacteria to begin destroying the dentin.

bacteria are responsible for both dental caries and periodontal diseases. Extension of these diseases commonly causes infection in the adjacent tissues, notably the pulp, periapical area and oro-facial soft tissues. More rarely, infection may become established in the bone of the jaw to cause osteomyelitis.

MAJOR FACTORS OF DENTAL CARIES.

1. Role of bacteria
2. Role of plaque:
3. Role of saliva:
4. Role of carbohydrates

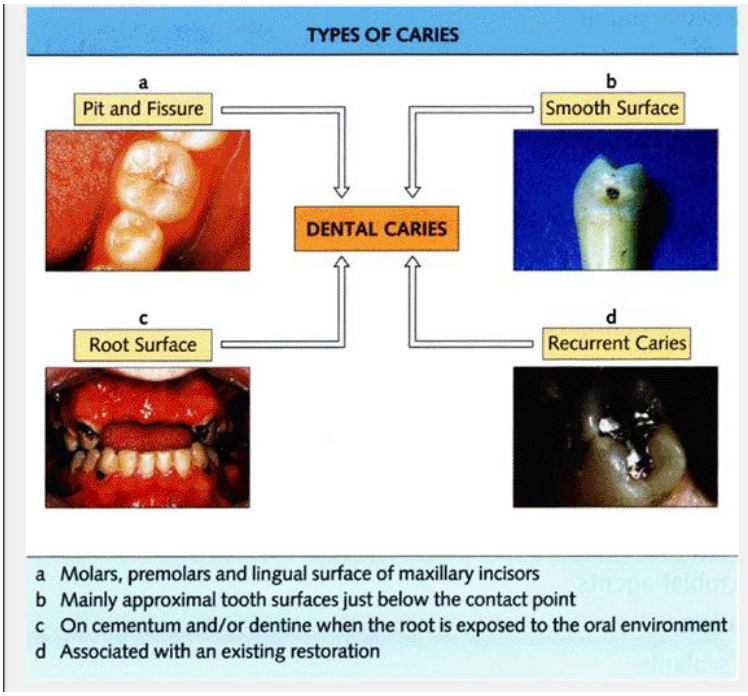
MINOR FACTORS:

- ◆ Enamel composition
- ◆ Morphology of the tooth
- ◆ Position of the tooth
- ◆ Diet
- ◆ Immunity .

CLASSIFICATION

Dental caries can be classified with respect to the site of the lesion (Fig. 32.1):

- ❖ pit or fissure caries (seen in molars, premolars and the lingual surface of maxillary incisors)
- ❖ smooth-surface caries (seen mainly on approximal tooth surfaces just below the contact point)
- ❖ root surface caries (seen on cementum or dentine when the root is exposed to the oral environment)
- ❖ recurrent caries (associated with an existing restoration).



Rampant caries can occur in particular subgroup of people who are especially prone to decay, such as xerostomic patients (who have a markedly reduced salivary flow rate due to radiation treatment for head and neck cancer), those with Sjogren's syndrome, or as a side-effect of medication.

'Nursing-bottle' caries is the extensive and rapid decay of the maxillary anterior teeth associated with the prolonged and frequent feeding of young infants with bottles or pacifiers containing formulas with a high concentration of fermentable carbohydrate. Plaque bacteria receive an almost continuous provision of substrates from which they can make acid.

CARIES PREVENTION

- Fissure sealants
- Fluoride (may also have some anti-glycolytic effects).
- Antimicrobial agents (these agents can be applied in toothpastes, mouthrinses and varnishes. Some also inhibit glycolysis).
- Sugar substitutes (prevent acid production, but xylitol may selectively inhibit Strep. mutans)

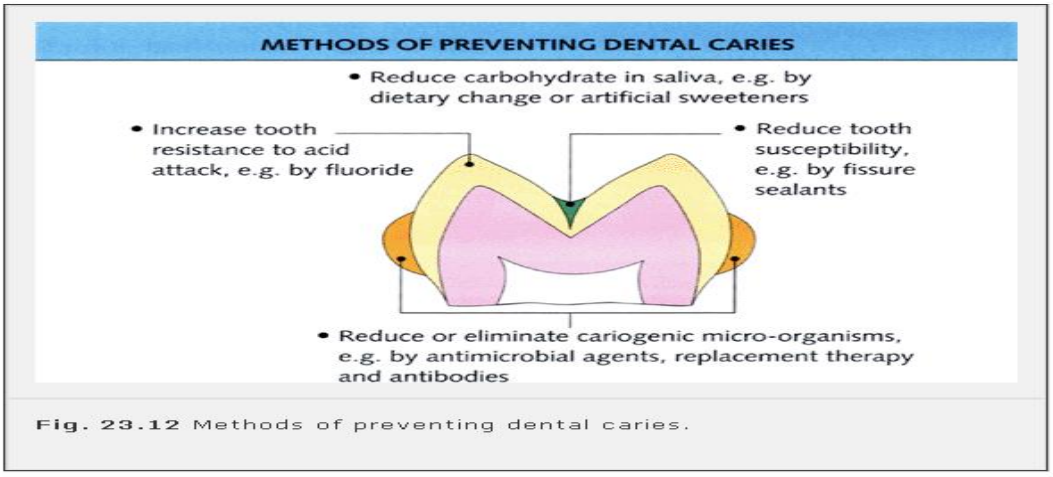


Fig. 23.12 Methods of preventing dental caries.

Treatment:-Treatment can help prevent tooth damage from leading to cavities.

Treatment may involve:

a-Fillings b-Crowns c- Root canals

<u>References:-</u>

1-Oral microbiology .5th edition.

2-Essentials of Microbiology for Dental Students. 2006 ,2nd Edition.