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The Effect of *Streptococcus Mutans* Bacteria on Tooth Decay

A project submitted
To the College of Dentistry, Misan University, in partial fulfillment of the requirements for the Degree of bachelor's in dental surgery (BDS).

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Certification of the Supervisor

I certify that this project entitled "**The Effect of Streptococcus Mutans Bacteria on Tooth Decay**" was prepared under my supervision at the College of Dentistry/University of Misan in partial fulfilment of the graduation requirements for the bachelor's degree in Dentistry.

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Date

Dedication

إلى من غرَسوا فيَّ بذور الحُلم، وسقوها بالصبر والدعاء،

إلى النورَين في حياتي، والديَّ العزيزين، الذين لولا دعمهما لما وصلت إلى هذا الموضع،

إلى كل مَن آمن بي، ووقف إلى جانبي حينما تزعزَعت الخطى،

إلى اساتذتي الأفاضل،

إلى أصدقائي وزملائي الذين كانوا العون والسند،

أُهدي هذا البحث... بكل فخر وامتنان

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بسم الله الرحمن الرحيم

(وَقُلِ ٱعْمَلُوا۟ فَسَيَرَى ٱللَّهُ عَمَلَكُمْ وَرَسُولُهُۥ وَٱلْمُؤْمِنُونَ)

صدق الله العلي العظيم.

الحمد لله الذي أنار دروب السائرين بنور العلم، وأسبغ على عباده نعمة العقل والتفكر، وبفضله وتوفيقه أنجزنا هذا البحث المتواضع.

أتقدم بخالص الشكر والتقدير إلى السيد عميد الكلية المحترم وأساتذة الكلية الأجلاء، الذين لم يدخروا جهدًا في تقديم العلم والمعرفة.

كما أخص بالذكر وأتوجه بوافر الشكر للأستاذة المشرفة

(م.م الضحى حسن عيسى) لما بذلته من جهدٍ مشكور في الإشراف العلمي والتوجيه الدقيق، حيث لم تبخل علينا بعلمها ونصائحها وملاحظاتها الدقيقة التي كان لها بالغ الأثر في إخراج هذا البحث بهذه الصورة.

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Abstract

Dental plaques are notorious and lead to dental caries responsible for dental decay. Streptococci are the leading microorganisms associated with dental plaques. These are Gram-positive, normal microbial flora, non- motile, non-spore forming, and facultative anaerobes. These include Alpha, Beta, and Gamma hemolytic species. *Streptococcus* sp. produces a high amount of lactic acid through the fermentation of sugars, causes lowering of the pH leading to the plaque formation around teeth and serves as a biofilm. Microbial biofilm provides certain attachment sites for growth and colonization of other bacteria and causes resistance to the antimicrobial agents. These Streptococci can be transmitted to the infants through parents or caretakers' kiss. This mode of transmission is the key role for the contribution of S. mutans in dental caries.[1]

Introduction

Dental caries is a process that may take place on any tooth surface in the oral cavity where dental plaque is allowed to develop over a period of time. Dental Plaque is an adherent deposit of bacteria and their products, which forms on tooth surfaces and is the cause of caries. Plaque is an example of a biofilm, which means it is not a haphazard collection of bacteria but a community of microorganisms attached to a surface. This community works together.[1]

The mouth contains a wide variety of oral bacteria, but only a few specific species of bacteria are believed to cause dental caries.

The most common bacteria associated with dental cavities are the mutans streptococci, most prominently Streptococcus mutans and Streptococcus sobrinus, and lactobacilli. However, cariogenic bacteria (the ones that can cause the disease) are present in dental plaque, but they are usually in too low concentrations to cause problems unless there is a shift in the balance. This is driven by local environmental change, such as frequent sugar intake or inadequate biofilm removal (tooth brushing). If left untreated, the disease can lead to pain, tooth loss and infection.[2]

Aim of study

The aim of this study is to investigate the role of *Streptococcus mutans* in the development and progression of dental caries, by examining its cariogenic properties, mechanisms of tooth enamel demineralization, and the conditions that promote its proliferation in the oral cavity.

Review of literature

1.1 Classification of dental caries

1.1.1 According to GV black

- Classified into 6 classes same as GV Black cavity classification.

1.1.2 According to surfaces involved

- Pit and fissures caries.
- Smooth surfaces caries.
- Root caries.

1.1.3 According to onset of occurrence

- Primary caries occur in previously intact tooth.
- Secondary (Recurrent) caries occur under previous restoration.[3]

1.1.4 According to caries progression

- Incipient (Initial) caries, not cross the DEJ with almost intact outer enamel surface and can be treated with remineralization, usually no restorative procedures required.
- Advanced caries, crossing the DEJ and restoration required.

1.1.5 According to severity of the lesion

- Acute or sever, fast progressing destructing the tooth extensively and often lead to pulp involvement. A term “Rampant” caries is usually used to describe acute caries affecting multiple teeth and it’s usually occur in children and adolescents
- Chronic caries, slowly progressing affecting particular tooth and pulp involvement is not as common as in active caries. **[3]**

1.1.6 According to activity of lesion

- Active caries mean that caries is in state of progression
- Arrested caries, mean that caries stop progressing and this usually occur if caries become in a self- cleansing areas (e.g. class II caries may be arrested after extraction of adjacent tooth.**[3]**

1.2 Dental plaque formation can be described in sequential stages:

- **Formation of pellicle:** an acellular, proteinaceous film, derived from saliva, which forms on a 'naked' tooth surface. .
- Within 0–4 hours, single bacterial cells colonize the pellicle. A large proportion of these are streptococci (S. sanguis, S. oralis, S. mitis). There are also Acintomyces species and Gram- negative bacteria. Only about 2% of the initial streptococci are mutans streptococci, and this is of interest because these organisms are particularly associated with the initiation of the carious Process **[1].**
- Over the next 4–24 hours the attached bacteria grow, leading to the formation of distinct **microcolonies**.
- In 1–14 days, the **Streptococcus-dominated plaque** changes to a plaque dominated by Actinomyces. Thus, the population shifts; this is called **microbial succession**. The bacterial species become more diverse and the microcolonies continue to grow. .
- In 2 weeks, the plaque is mature but there are considerable site-to-site variations in its composition. Each site can be considered as unique and these local variations may explain why lesions progress in some sites but not others in the same mouth.**[1]**

1.3 Causes of dental caries

There are four main criteria required for caries formation: a tooth surface (enamel or dentin); cariogenic (or potentially caries causing) bacteria, fermentable carbohydrates (such as sucrose); and time. The caries process does not have an inevitable outcome, and different individuals will be susceptible to different degrees depending on the shape of their teeth, oral hygiene habits, and the buffering capacity of their saliva. Dental caries can occur on any surface of a tooth that is exposed to the oral cavity, but not the structures, which are retained within the bone.**[4]**

1.4 Environment of the tooth

1.4.1 Saliva and fluoride

Under normal conditions, the tooth is continually bathed in saliva. Saliva is supersaturated with calcium and phosphate ions and capable of remineralizing the very early stages of lesion formation, particularly when the fluoride ion is present. Fluoride slows down the progression of lesions.When salivary flow is diminished or absent, there is increased food retention. Since salivary buffering capacity has been lost, an acid environment is encouraged and persists longer. This in turn encourages acid uric bacteria, which relish the acid conditions and continue to metabolize carbohydrate in the low-pH environment. The stage is set for uncontrolled carious attack.**[1]**

1.4.2 Normal Flora of the Oral Cavity

The presence of nutrients, epithelial debris, and secretions makes the mouth a favorable habitat for a great variety of bacteria. Oral bacteria include streptococci, lactobacilli, staphylococci and corynebacteria, with a great number of anaerobes, especially bacteroides. The mouth presents a succession of different ecological situations with age, and this corresponds with changes in the

composition of the normal flora. At birth, the oral cavity is composed solely of the soft tissues of the lips, cheeks, tongue and palate, which are kept moist by the secretions of the salivary glands. At birth the oral cavity is sterile but rapidly becomes colonized from the environment, particularly from the mother in the first feeding. *Streptococcus salivarius* is dominant and may make up 98% of the total oral flora until the appearance of the teeth (6 - 9 months in humans). The eruption of the teeth during the first year leads to colonization by *S. mutans* and *S. sanguis*. These bacteria require a nondesquamating (nonepithelial) surface in order to colonize. They will persist as long as teeth remain. The normal bacterial flora of the oral cavity clearly benefit from their host who provides nutrients and habitat.**[4] [1]**

1.4.3 Natural resistance of oral microflora

Natural resistance of bacteria also called intrinsic resistance could be attributed to the absence of particular structures and\or lack of specific metabolic pathways that are crucial for the activity of the antibacterial agents. In oral microflora, natural resistance is attributable to the absence of specific metabolic processes. For instance, the enzyme nitro- reductase, which is essential for the conversion of metronidazole to its metabolic products, is not present in Actinomyces spp., Streptococcus spp. and Aggregatibacter sp. Besra and Kumar, (2016) added that the activity of metronidazole does not harm these microorganisms at regular therapeutic meditations. **[1]**

1.4.4 Acquired resistance of oral microflora

Compared to the mechanism of natural resistance, acquired resistance is resent in certain bacterial species due to genetic variation, and can be attained by two primary mechanisms mainly:

- Chromosomal alteration in the established bacterial genome.

- Horizontal gene transfer between the bacterial species.
- According to Daboor et al., (2015), the horizontal gene transfer mechanism (most frequent pathway) permits the bacterial inhabitants to develop acquired resistance against the antibiotics at a higher level, which is considerably greater than that would be afforded by the chromosomal alterations. A recent study of Childers et al., (2017) revealed that due to the low frequency of side effects, low-cost effectiveness and appropriate antibacterial spectrum, penicillin group of antibiotics have long been used as the first line of defence against the dental diseases. These conventional antibiotics can interfere with dental biofilm development. Additionally, penicillin antibiotics are not suitable for prolonged use, due to the development of resistance and ecological disparity favoring the adaptable dental diseases [1]

1.5 Common Characteristic Features of Species of Mutans Streptococci

*S. mutan*s is Gram-positive cocci in pairs and chains, Facultative anaerobe Alpha hemolytic or non-hemolytic on blood agar. On sucrose containing agar, produce extracellular polysaccharides. Extracellular polysaccharides may be water-soluble glucans and fructans or water insoluble glucan and fructans.

Appearance of colonies: Colonies are frequently white, rough, heaped and detachable. A drop of liquid (water-soluble glucan) may appear on the top of the colonies. Produce acid from a wide range of carbohydrates, viz. amygdalin, lactose, maltose, sorbitol, etc.

Acid is not produced from starch, erythritol, etc. Alkaline phosphatase and urease not produced. Three polysaccharide antigen c, e, f present (k is also recognized). Cell wall peptidoglycan is Lysine-Alanine. The various strains of *Streptococcus mutans*, their source, serotypes, sugar Fermentation. [4]

1.6 Pathogenicity and Virulence of Streptococcus Mutans

S. mutans are cariogenic organisms, residing in the human mouth and occasionally causing dental caries. Because it is commensal, it has a different mechanism that allows it to adhere to and colonize the musical membrane of the oral cavity ;it can also be passed from one person to another via horizontal and vertical transmission. S. mutans is most frequently transmitted to infant children from their mothers،the vertical transmission of S. mutans can be detected if the organism is found in the furrows of the tongues.

Accordingly 'pathogenicity' of a given microorganism is expressed by the degree of damage caused by the micro-organism itself and also by the immune system in response to the pathogen.

Pathogenicity is due to:

- Invasiveness (the ability to invade the host tissues).
- Toxin production (substances that damage and/or kill cells). (However, some bacteria and the viruses do not produce toxins, but kill/damage cells by their replication.)

Virulence' on the other hand is the ability of microorganism to cause infection; dependent on the degree of pathogenicity. The virulence of a microorganism is determined by the following:

- Division rate.
- Quality and quantity of toxins produced.
- Speed of invasion.
- Body's resistance (immune system).
- Cellular features, such as motility, attachment, etc.

Usually two types of toxins are recognized:

- **Exotoxins** produced inside mostly by gram-positive bacteria as part of their growth and metabolism they are then release into surrounding medium

- **Endotoxins** released after the death of bacterial cells; mainly produced by gram-negative bacteria. The virulence factors of streptococci mutans are surface proteins, acid tolerance, acid production and production of glucosyltrans-ferases, mutacin and intracellular polysaccharides. Many oral streptococci do produce sIgA protease, which impairs the host defence. Another important factor is the ability of mutans streptococci to rapidly adapt to the environment by microbial genetic phenomena (because of this property, Streptococcus mutans dominated in cariogenic dental plaque).**[7]**

Following factors contribute to virulence characteristics of Streptococcus mutans:

- Adherences to tooth surfaces and other bacteria.
- Rapidly metabolizing nutrients.
- Acidogenicity and acid tolerance (aciduric).
- Mutacin production. **[8]**

1.6.1 Adherences to Tooth Surfaces and other Bacteria

Streptococcus mutans have the potential to convert sucrose to glucan and dextran. The adhesion of S. mutans to dental plaque is mediated via sucrose-independent and sucrose-dependent means. Even though sucrose-independent adhesion to the acquired enamel pellicle might initiate the attachment process, but sucrose-dependent adhesion is primarily responsible for establishing colonization to the tooth surface.

These streptococci produce large amounts of extracellular dextran, a glucose-containing polysaccharide, whereas the polysaccharides produced by the non-cariogenic strains were primarily of fructose-containing type.

Bacterial adhesion may modulate susceptibility and resistance to dental caries. Adhesion of Streptococcus mutans correlates with high caries experience

Another potentially pathogenic factor is the ability of micro-organisms to synthesize and degrade intracellular polysaccharides (function by which the organisms continue fermentation and produce acid in the absence of exogenous carbohydrate). The synthesis and degradation of intracellular polysaccharides may play an important role in the ability of various bacteria in initiation and progress of caries.**[15]**

Bacterial proteins that help in adherence are:

- Antigen I/II family.
- Adhesin.
- Fimbrial adhesion.
- Glucan binding protein.

Initial attachment of Streptococcus mutans to tooth pellicle is mediated by antigen I/II (adhesin) and Glycosyl transferase (Gtf). Agglutinins present in saliva contribute to the process of Streptococcus mutans adhesion. Sucrose is broken down into fructose and glucose by glycosyltransferases. Fructose and glucose are metabolized, resulting in lactic acid formation, which accumulate over the surface. Glucose is stored as a glucan polymer (dextran). Streptococcus mutans adhere to glucans produced by other bacteria in plaque. **[9] [14]**

1.6.2 Rapidly Metabolizing Nutrients

Streptococcus mutans utilize dietary sucrose to enhance colonization of the oral cavity. The quantity of this organism in the oral cavity can be increased/reduced by increasing/decreasing the dietary intake of sucrose. Streptococcus mutans act on sucrose resulting in production of glucans/levans, which results in plaque formation, and acids, which results in demineralization of tooth structure. Sucrose-6-glucosyltransferase dextran sucrose, an enzyme produced by Streptococcus mutans converts sucrose to dextran. Dextran has the

property of causing clumping of bacteria producing their aggregates. Oral microorganisms derive nutrients from saliva and gingival crevicular fluid. Additionally, exogenous substrates are provided intermittently in the diet. Thus, there is enormous diversity in substrates available and in the metabolic activities of the organisms, which colonize the oral cavity. Carbohydrates are metabolized by multiple sugar metabolism (MSM) system present in bacterial cytoplasm. These are transported via the phosphoenolpyruvate (PEP), an important enzyme in this pathway. Fluorides inhibit the functioning of this enzyme (emphasizing the role of fluorides in caries prevention). Glucosyltransferases (Gtfs) and fructosyl-transferases (Ftfs) catalyse the synthesis of water- soluble and water-insoluble glucan and fructan polymers from sucrose. **[12]**

1.6.3 Acidogenicity and Acid Tolerance (aciduric)

It is established that pH below 5.5 (critical pH) results in the dissolution of calcium phosphate (hydroxyapatite) of the tooth enamel. Promotion in the growth of aciduric bacteria further lowers the pH and promotes progression of the carious lesion.

The mutans streptococci metabolize sucrose to lactic acid more rapidly than other oral bacteria. This might be related to the enzyme systems catalysing the metabolism of sucrose. These metabolic reactions render the dental plaque acidic and the mutans streptococci continue metabolisms even at low pH beyond the salivary buffering capacities It has been established that streptococci mutans are more acid tolerant than other oral bacteria, with the exception of lactobacilli. The property of acid tolerance (or acidurance) is related with the membrane's H+ (proton)-translocating adenosine triphosphatase (ATPase) of these organisms. And involves adaptation with a resulting change in gene and protein expression. Together they constitute the acid-tolerance response (ATR).**[10] [13]**

1.6.4 Mutacin production

Many bacteria produce bacteriocins (an antibacterial peptide), which interfere with the growth of other microorganisms. The genes involved in the synthesis and modification of bacteriocins are often carried by a plasmid. Bacteriocins are frequently named according to the bacterial species producing them, such as bacteriocin produced by mutans streptococci is called mutacin. Mutacin production is usually not plasmid encoded. If bacteriocin activity is plasmid encoded, it confers bacteriocin immunity to the microorganism. It is documented that strains producing increased amounts of mutacin colonize more easily**.[11]**

1.7 Relation of Streptococci to Caries

Oral streptococci are considered as the main causative agents in carious process. The *Streptococcus mutans* have the maximum cariogenic potential and is described as follows:

1.7.1 Primary Acquisition and Transmission of *Streptococcus mutans*

The inoculation of the human oral cavity starts immediately after the tactile contact of child with the environment. The oral cavity of the toothless child contains only epithelial surfaces and the first colonizers are species, which do not require a non-shedding surface for their survival. Early colonizers include streptococci, Veillonella, Acintomyces, Fusobacterium and also a few gram-negative rods. Amongst them, Streptococcus salivarius mostly colonize the dorsum of the tongue. Streptococcus sanguis and the Streptococcus mutans are colonized only after the first tooth has erupted. The acquisition of micro-organisms by the human body is by direct transmission from one host to another, or through some objects. Pathogens can also be transmitted by food and water.

Saliva is regarded as the most important vehicle for transmission of mutans streptococci via physical contact. The mother is considered to be the most important source of infection for the child. The time period when children gain mutans streptococci in their oral flora is when the primary teeth are erupting, that is, between 6 months and 30 months of age. The probability of colonization with mutans streptococci is high when inoculation with mutans streptococci is frequent (the baby's diet includes frequent intake of refined carbohydrates).Oral streptococci have the cariogenic potential and the feature which mainly relates to cariogenicity is that their rate of growth and acid production exceeds that of any other oral micro-organisms.**[6]**

1.7.2 The other features supporting their role as cariogenic organisms are:

- Rapid generation time as compared to other oral bacteria living in the same environment.
- Acid producing qualities (terminal pH4), indicating high Acidogenicity.
- Ability to attain the critical pH required for enamel demineralization
- More rapidly than other oral bacteria.
- Facilitate fermentation of carbohydrates available in human diet.
- Facilitate plaque formation with the help of fermentable carbohydrates.
- Ability to initiate and maintain microbial growth and continue acid production at low pH.
- Production of extracellular polysaccharides from sucrose.
- Significantly correlates with progression of carious lesion.
- Significantly correlates with incidence/prevalence of caries.

Effective in experimental caries in animals. (Immunization of animals with Streptococcus mutans significantly reduces incidence of caries.)**[7][6]**

1.8 Prevention of Dental Caries

Streptococcus mutans possesses the abilities to adhere to pellicle-coated tooth surfaces and to form acids - two characteristics associated with the cariogenicity of this microorganism. De novo synthesis of insoluble glucan by S. mutans glucosyltransferase from sucrose is essential in the adherence process. Therefore, agents which interfere with the adherence ability of S. mutans would be useful for controlling dental caries**.[16]**

Preventive measures in mothers influence the establishment of the bacterium Streptococcus mutans in their infants

First-time mothers who had a high salivary number of Strep. mutans [≥ 106 colony-forming-units (c.f.u.) per ml were selected. Every second mother was given a special preventive programme to reduce her salivary level below 3 × 105 c.f.u. per ml. Where a reduction of Strep. Mutans was achieved in the mother, the establishment of Strep. mutans in her infant was prevented or delayed. Thus, 28 mothers were successfully treated until their infants were 23 months old and only 3 of their infants (11 per cent) were infected with Strep. mutans, compared with 17 out of 38 infants in the control group (45 per cent). In both groups, the percentage of infected infants increased with increasing age, although at all ages fewer infants were infected with Strep. mutans in the test group than in the control group. Sixteen infants of successfully treated mothers had reached the age of 36 months. Three were infected (19 per cent) compared with 17 out of 27 in the control group (63 per cent). These findings show that the spread of Strep. mutans can be delayed or prevented by measures directed against the main source of infection, an approach which is successful in the prevention of other infectious diseases**.[17][18]**

1.8.1 General Guidelines for Preventive Regime

Following guidelines should be implemented in any preventive regime:

- Basic oral health care information should be provided to each individual. 2-Motivation as regard to awareness of importance of prevention.
- Good oral hygiene as an essential part of the general body hygiene should be emphasized.
- Use of fluoride containing toothpastes (wherever required).
- An adequate non-cariogenic diet should be recommended.
- Use of other preventive measures.
- The guidelines should orient towards health promotion, health awareness, self-care and self- reliance.**[5]**

1.8.2 Preventive care regime

The preventive care regime is divided into four levels:

- **Care level 1:** Aims at increasing awareness of oral health, educating and motivating the individuals to adopt preventive measures.
- **Care level 2:** Includes informing the people about the available preventive aids and their clinical use.
- **Care level 3:** Includes initial restorative procedures and removal of the etiologic factors.
- **Care level 4:** Deals with tertiary preventive regimes**.[5]**

1.9 Control of caries inducing microorganism

It has been established that the plaque formation precedes the caries process. Plaque consists of a bacterial film that produces acids as a by-product of its metabolism. These acids have the potential to dissolve calcium and phosphate contents of the tooth substance; the process known as demineralization. The process of demineralization can be reversed (i.e. remineralization); if not, will lead to initiation of caries.

The deteriorative effect of dental plaque accumulation and metabolism is bidirectional, i.e. outward towards soft tissue causing periodontal disease and inward towards the tooth causing dental caries.

WHO defined dental plaque as 'a specific but highly variable structural entity resulting from sequential colonization and growth of micro-organisms on the surfaces of teeth and restorations consisting of micro-organisms of various strains and species embedded in the extracellular matrix, composed of bacterial metabolic products and substances from serum, saliva and blood.'

Plaque control measures are considered as the prime approach to prevent dental caries. It includes mechanical procedures as well as chemical agents, which retard plaque formation. Mechanical plaque control is indispensable to achieve optimum success and chemical measures are used only as an adjunct to mechanical means and not as a substitute.

The procedures of personal plaque removal by tooth brushing and/or flossing help maintain good oral hygiene. In addition, tooth-brushing is a proven caries preventive procedure, especially with the self-application of fluoride dentifrice. The caries control protocol includes thorough plaque removal, use of fluorides and dietary counselling. Professional tooth cleaning also has an important effect on caries reduction. Effective plaque removal reduces the development of new carious lesions**.[5]**

The guidelines as regard to maintaining oral hygiene vis-à-vis preventing caries are as follows:

- Maintaining oral hygiene requires a lot of motivation. Instructions should include the available mechanical aids and their selection, based on the patient's needs.
- Routine brushing with approved dentifrices.
- Thorough rinsing and flossing after every meal.

- During sleep, a decrease in salivary flow allows unrestricted plaque growth. Therefore, emphasize oral hygiene measures before bedtime.
- The routine flossing removes debris and bacterial plaque from the interproximal spaces.
- Rinsing should follow flossing and brushing. Rinsing is repeated until the expectorated rinse water is clear**.[5]**

1.10 Methods of Plaque Removal

The plaque removal methods are categorized into following:

- Mechanical.
- Chemical.
- Miscellaneous. **[5]**

1.10.1 Mechanical Plaque Control

The mechanical plaque control devices include:

a) Toothbrushes.

- Manual.
- Powered.

b) Dentifrices.

c) Auxiliary aids/interdental aids.

- Dental floss.
- Wooden/plastic sticks.
- Interdental stimulators.
- Interdental brushes.
- Dental tape.

d) Oral irrigation.

Oral irrigation with different solutions is an effective aid for removing debris from the inaccessible areas like interdental areas, pockets, furcation and

around orthodontic appliances and fixed prostheses.Various irrigating solutions, viz. warm water, normal saline, iodine solutions, sanguinarine, essential oils, chlorhexidine, etc. have been used. A few authors observed transient bacteremia with water irrigation; however, it is considered an easy and safe irrigant. **[5]**

1.10.2 Chemical Plaque Control

Mechanical measures are the primary methods for removal of debris and plaque. Chemical agents as plaque inhibitors have also been tried; however, these agents should not replace the mechanical measures. The chemical agents are used as an adjunct to mechanical techniques and should be prescribed according to the needs of the individual. The requisites of antiplaque agents are:

- Should eliminate the pathogenic bacteria only.
- Should not develop resistance.
- Substantively (the agent should remain in contact with the surface for a longer period).
- Safety to the oral tissues at the recommended concentration and dosages.
- Should not stain the teeth or alter the taste.
- Easy to use.
- Inhibits calcification of plaque.
- No adverse effect on teeth and restorations.
- Cost effective.**[5]**

The commonly used chemical plaque control agents are:

- Chlorhexidine (Bisbiguanides).
- Essential oil (Listerine).
- Delmopinol (Decapinol).
- Fluorides.
- Metal ions.

- Iodine.
- Iodophors.
- Chloroxylenol.
- Natural products.
- Enzymes.
- Triclosan.
- Oxygenating agents.
- Quaternary ammonium compounds.
- Sodium benzoate.
- Antibiotics. **[5]**

Conclusion

The main virulence factors of S. mutans are its ability to utilize sucrose to promote adhesion and accumulation in dental biofilms, its acidogenicity and its tolerance to acids. The acid survival of S. mutans depends both on the pH of the medium and on the composition of fatty acids and proteins plasma membrane (F-ATPase and P-ATPase).

As with most host–microbe interactions, these attributes only provide the organism with pathogen potential. The physiology of the host and the overall oral flora ecology may or may not suppress this potential.

The advance in the knowledge of how complex and heterogeneous can be the disease of the caries, according to the surface or the biofilms where it develops, can be useful to design new strategies of therapy in the treatment of this disease. The oral environment also plays a vital role in banking the organism.

However, the tolerance level varies from one individual to the other and it's also depends on the molecular entities in the organism that enables them to metabolize appropriate substrate leading to a convenient environment which could have a negative influence on the individual's health as well as the oral microflora.

Sucrose has been disclosed as one of the main substrates for the organism to survive but lactose has also been highlighted by certain studies.

However further analysis is required to have a deep-seated comprehension on facets like microbial interaction within the biofilms and the prominence of molecular entities.

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