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The Role of *Streptococcus mutans* and Pathogenesis in the Oral cavity

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Abstract

The oral cavity is a complex environment system where certain bacteria coexist with human chemical compounds. However, changes in the natural structure of the bacteria may lead to the onset of oral disease, such as periodontitis and tooth decay. A number of different environments exist in the human oral cavity, colonized by more than 600 types of aerobic and anaerobic bacteria, including the teeth, gingival sulcus, hard and soft palates, and tonsils, but only a limited number of these types can cause tooth infection .

The purpose from this study is an attempt to establish which characteristics associated with biofilm formation—virulence determinants of *S. mutans*—are responsible for the development of dental caries

The review concluded the bacterial components that contribute to each of the major virulence properties and these are work together in the development of dental caries .

Keywords: *Streptococcus mutans* , Dental caries , Pathogenesis

Oral streptococci

Oral streptococci are classified into four species groups; the anginosus, mitis, salivarius, and mutans groups [1] .Classification According to chemotensive and genotypic data, especially the DNA base pairing and 16-ribase gene sequence analysis, the mutant group include *S. mutans* , *S. sobrinus, S. downeii, S. rattus, S. macacae s* and *S.cricetus*, .[2].

It is Belongs to the Streptococcus group Viridans (*S.mutans, S.sanguis, S.salivarius, S.mitis*) [3] .The streptococcus mutant consists of *S.mutans, S. Rati, and S. Sobrinus* is found in a large number on the surface of the teeth [4] ,while *S..salivarius* is isolated mainly from the tongue one of the main alkali producers in the mouth [5]. *Streptococcus sanguinis* is a member of the human indigenous oral microbiota

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It is known as a pioneering colonizer in the formation of dental plaque and it has an antagonistic activity against *S. mutans* [6]. *Streptococcus.mitis* has traditionally been considered as harmless living in the pharynx [7].

Streptococcus mutans

Streptococcus mutans is a member of the micro flora, It is considered to be the main causative agent for tooth decay and one of the best biofilm formation [8]

S. mutans binds to the biofilm on the dental surface by converting sucrose into extracellular polysaccharide adhesive substance (Dextran), but when in existent the sucrose in the mouth, it was produce the dextranase enzyme that splits the dextran matrix of dental plaque as carbon source due to produce of lactic acid that demineralized tooth enamel[9].

The etiology of tooth decay are associated with the production of organic acids resulting from bacterial fermentation of dietary carbohydrates,their ability to produce glucosyl-transferase (GTFS), which leads to the formation of intracellular polysaccharides (IPS), extracellular polyaccharides (EPS) ,its coherence on the surface of the tooth enamel by glucan [10] . The combination of these feature mechanisms is acquired by *S. mutans* its effective colonization in the mouth and organizes the transformation from non-pathogenic to cariogenic dental plaque biofilm [11] .

S. mutans are the most common species isolated from human saliva and play fundamental role in the development and progression of tooth deacy [12]. Among the physiological traits of mutans streptococci which are most relevant to cariogenesis are their synthesis of extracellular polysaccharides from sucrose which fosters their firm attachment to teeth and promotes tight cell clustering, rapid fermentation of carbohydrates to acids and tolerance to low pH.

It has been proven that mutans streptococci can colonize the mouth of infants before dentate are obtained through vertical and horizontal transition from both human reservoirs, especially mothers . Earlier in childhood occurring mutant charges high salivary streptococcus, and more severe caries in primary teeth [13]

Mutant streptococci also show significantly higher prevalence of streptococci and higher percentages in positive caries subjects than individuals free from caries. [14].

Among mutant streptococci, *S. mutans* has often been associated with the onset and development of dental caries and is generally considered as a major factor for human tooth decay [15].

Mode of Transmission of *S. mutans*

Since the absence of oral cavity is sterile, the child first becomes colonized by a normal flora at the instant of passage through the birth canal. *S.mutans* cannot be detected in human newborns and colonization occurs early in life, even before the first age eruption [16].

Saliva is the most common means of transmission of *S. mutans* through physical contact. This substance is the most important source of infection for the child [17]. (

Children usually receive *S.mutans* from their mother (vertical transmission) or (horizontal transmission) from another child, the most common transmission mode is the transmission from the mother to the child, the first days of life and feeding by the mother during birth, some microorganisms . *S. mutans* colonize the oral cavity produce complex microbial community after complete eruption of teeth are acquired during a primary "window of infectivity" nineteen months of age, immune status and the extent of sucrose intake might influence the initial dental caries

Numerous studies reported similar mutans streptococci genotype identification which are common to mother, father and child [18] .

Also transmission of infectivity from age group to another, through the fluid such as saliva or the placenta or through milk [19].The second "infection window" has also been proposed in 6-12 years of age and ends with a permanent tooth eruption, clonal distribution suggested that the mother is the most common source of infection in children with MS. However, detection of genetic patterns in children not found in their mothers or relatives indicates other sources of transport, many evidences have been reported of horizontal transmission between children at the same nursery and older [20]. Horizontal transmission can also occur between family members. It is often spread through contaminated sputum, saliva, blood from person to person [21].

Characterization and Identification of *S.mutans*

S. mutans is a Gram-positive, oval-shaped spherical shape, organized in pairs or short chains during the growth of bacteria, non-spore formation, facultative anaerobic, non-motile - It is a synthesis of the dextran polysaccharide capsule, the catalase-negative [22] , Alpha hemolysis is usually on blood agar plates. *S.mutans* is mesophilic and grow at temperatures between 18-40 C^0 [23] , Their colonies appear in light blue (1-2 mm) in diameter in an oval or spherical shape in shape with a convex surface of MSB agar [24].

Biochemically, *S.mutans* strains give oxidase, motility, Simmon's citrate , and urease negative [25], It is able to ferment glucose and many types of sugars such as sucrose, ribose, sorbitol, mannitol, fructose and lactose [26].

The mutant streptococcus is mostly found in the oral cavity, a major contributor to tooth decay. [27] . It has virulence factors such as: glycans insoluble water, acid tolerance, and lactic acid production [28].

Dental caries

Dental caries is still one of the most common chronic diseases with bacterial food pathogens[29].Characterized by decay before the environmental transformation within the biofilm dental environment, driven by frequent access to fermentable dietary carbohydrates, leads to a move from a balanced population of microorganisms of low cariogenicity to a microbiological population of high cariogenic and to an increased production of organic acids .[29]. .

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Dental caries occurs due to dental plaque (biofilm) lying on the teeth and maturation to become cariogenic (causing caries). Some bacteria produce biofilm in the presence of carbohydrate fermentation such as sucrose, fructose and glucose. [30].

The *S. mutans* was the only bacterium found in significantly larger numbers in the carious lesions than on the teeth of caries-free individuals [31]. Molecular analysis of bacterial samples attained from the enamel of caries-free children and from within the carious lesions of children suffering from Early Childhood Caries, showed a similar cause-and-effect pattern.[32].

Pathogenesis of *S.mutans*

Pathogenicity is the ability of microorganisms to produce disease in a host organism. Microorganisms express their morbidity through virulence [33], The disease probably occurs even more. These factors include I-Invasiveness a microbial ability to invade tissue. It includes mechanisms for colonization (accession and initial reproduction), extracellular production, which facilitates invasion and the ability to be evaded by host defense. II-Toxigenesis, which is the ability to synthesize toxins [34].

The *S. mutans* is the main causative agent for human tooth decay [35]. A difference in mechanisms has been developed to colonize the tooth surface. These bacteria acquire the ability to search for multiple sugars that are fermented to grow and to dissolve sugar in low pH values in the mouth. Acid production by *S.mutans* causes the dissolution of minerals on the surface of the teeth and the formation of tooth decay, [36].

Pathogenicity of *S. mutans* plays three important roles. First, the ability to form a biofilm. In general, the formation of biofilms is a two-step sequential process that requires binding the bacterial cells to the surface (early adhesion), followed by accumulation of growth-dependent bacteria in several groups of multilayered cells, and the two-stage mechanism attached to the initial. Formation of biofilm by *S. mutans* mechanisms independent of sucrose and dependent sucrose [37].

The second feature is the ability of bacteria to produce acids (acidogenesis) [38] and the third important characteristic is the ability to survive in pH-tolerant environment (aciduricity) [39].

Fimbriae factors are called adherence that act as adhesion the bacteria in epithelial cells. The number of bacterial members has a parasite that responds to the adherence to the surface of the epithelial cells, different strains or types of bacteria may produce different types of fimbriae [40] , which can be characterized on the basis of morphology, antigenic composition and receptor specificity. A broad division is made between fimbriae in which adherence in vitro is affected by D-mannose (mannose – sensitive fimbriae) and those unaffected by this treatment (mannose–resistant fimbriae). The antigenic composition of fimbriae can be complex, which can be described on the basis of morphology, antigen composition and future specificity.

S. mutans is able to producing biofilm, and the development of biofilm occurs in two stages. Distinction: During the first stage , bacterial surface proteins interact with the host or colonized bacterial products on the enamel. In the second stage, the biofilm

forms for the bacteria accumulate by aggregation with the same or other species and production Extracellular glucan [41]. .

An important feature of *S. mutans* in promoting caries development is the ability to strongly adhere to enamel teeth in the presence of sucrose [42] .The difference texture in insoluble water tissue - glucan (WIG) or biofilm formation between the genotype with different levels of virulence. This is important, since it has been demonstrated that WIG generated from sucrose modifies the dental plaque's physiochemical properties, including decreased concentrations of phosphate, calcium and fluoride and high porosity of the dental plaque matrix [43]. (

The main mechanism of adhesion dependent on sucrose is action (GTFs) in the synthesis of glucan. GTFs have sucrose activity, which produces in cleavage sucrose, to glucose and fructose [44] .The glucose partion is then added to a growing polymer of glucan.

The *S. mutans* contains three GTFs encoded by gtfB, gtfC, and gtfD contains three genes encoding GTFs. Aggregatively, the GTFs synthesize both water-soluble and WIGs. The water- soluble glucan (WSG) is

prevalently linear polymer linked by alpha 1,6-glycosidic linkages that called dextran, the WIG is prevalently a higher degree of branching polymer linked by 1,3-linkages that called mutan. Both polymers are thought to participate to sucrose dependent colonization and decay, but WIG may be of primary importance for smooth surface decay [45]. .(

The *S. mutans* include 1,500 GTFs of long amino acid and possess two functional domains, the absolute amino fraction is the splitting of the catalyst from the peripheral part of the sucrose and carboxyl which stimulates the dextran binding [46]. Numerous studies have shown "using GTF mutant strains of *S. mutans*" decrease in the cariogenicity of bacteria and the ability to adhesion to smooth surfaces or from biofilm in vitro [47 and 48].

Conclusion

The *Streptococcus mutans* is a potent initiator of caries because there is a variety of virulence factors unique to the bacterium that have been isolated that play an important role in caries formation . Dental caries is a seemingly simple problem with complicated underlying causes, which will hopefully be resolved through genetic engineering, preventative techniques, and other treatment options based upon an evolutionary understanding of our relationship with *Streptococcus mutans*.

Conflict of Interests.
There are non-conflicts of interest .

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