Microbes and Infectious Diseases 2025; 6(1): 171-183

Microbes and Infectious Diseases

Journal homepage: https://mid.journals.ekb.eg/

Review article

Streptococcus mutans - The life on human teeth –An extensive review on molecular mechanisms and consequences for systemic health

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ARTICLE INFO

Article history:
Received 29 January 2024
Received in revised form 27 February 2024
Accepted 9 March 2024

Keywords:
Streptococcus mutans
Dental caries
Quorum sensing
Biofilm
ComABCDE pathway

ABSTRACT

Background: *Streptococcus mutans*, a bacterium commonly found in the oral cavity, displays remarkable adaptive capabilities that enable its survival in this unique environment. Maintaining good oral hygiene practices, adhering to hygienic behaviors, and adopting healthy dietary habits have been identified as potential factors for restraining the growth of cariogenic bacteria. While numerous strategies have been developed for managing dental caries, the ultimate goal is to identify preventive agents that can effectively halt the progression of caries and impede the development of new lesions. This review aims to explore the competent behavior of *Streptococcus mutans*, specifically its role in causing oral infections that can have prolonged effects on systemic health. The establishment, persistence, and strengthening of the interaction between species response and the human system contribute to this phenomenon. The implications of *Streptococcus mutans* in human health are significant, and as such, this review focuses extensively on its putative virulence factors, the mechanisms by which it exerts its virulence, and the clinical complications associated with its presence. By providing a comprehensive understanding of the molecular mechanisms employed by *Streptococcus mutans* and the consequences of its colonization in the oral cavity, this review expands our knowledge of the potential systemic implications associated with this bacterium. Ultimately, it calls for the development of targeted preventive strategies to mitigate the negative impact of *Streptococcus mutans* on both oral and systemic health.

Introduction

Human body forms an anchoring habitation for assorted group of microorganisms in which the oral cavity conquers the prodigious space both in health and diseased condition. The human body shares their body space with those microbial communities and form a symbiotic, commensal and pathogenic relationship with each other since it provides a highly diverse and site-specific microenvironment [1]. The vast range of bacterial communities in the oral environment is collectively called as oral microbiome. The oral microbiome is mainly imperative to health because it can cause both oral and systemic diseases. The association of diseased symptom can be correlated between the patients, in such case there exist variability in

DOI: 10.21608/MID.2024.266348.1781
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microbial community composition that undergoes conserved changes in metabolism during disease [2].

There are numerous microorganisms associated with the oral disease in which *Streptococcus mutans* is a relatively super-prime organism that encounters to have an effective site than the neighbouring organism because it forms an obstructed environment for the relative microorganisms [3]. This engrossing pathogen was first described by Clarke in 1924. Clarke associated *Streptococcus mutans* with human decay, but other investigators were unable to find *Streptococcus mutans* and this organism eventually became a nonentity. It was rediscovered in the 1960s as investigators sought to identify the *Streptococcus* shown to cause a transmissible infection in the rodent models. Once investigators sampled plaques from single carious sites or saliva from caries-active individuals, *Streptococcus mutans* was routinely associated with human dental decay [4].

There are several infections occurring in the oral cavity which correlate with the efficacy of the oral microflora for the development of dental caries, periodontitis, rampant caries, xerostomia, diabetes, cardiovascular disease. This order of disease progression starts from the opening of the oral cavity to the interior of the heart region [5].

Streptococcus mutans links up with the acquired enamel pellicle (AEP) and associative microorganism through a combination of sequential path and causes dental caries, in humans. An accumulation occurs by the intake of sucrose by human beings in their diet and the pre-existing primary colonizers utilize the sugar substrate by glucosyltransferases (GTFs) which then produces glucans whereas it binds with their specific glucan binding proteins (GBPs) to make anchorage on the dental enamel and further demineralize the hydroxyapatite (HA) deposition layer of the tooth surface by their liberated acid by-product. Assemblage of these characteristic mechanisms is acquired by *Streptococcus mutans* which aids its effective colonization in the oral cavity and regulate the transformation from non-pathogenic to extremely cariogenic dental plaque biofilms [6].

Though there are several factors are responsible for the disease development on such crucial element is the colonization of microbial community by cell-to-cell communication. The factor of genetic competence to colonize the hard tooth surface is facilitated by Competence Stimulating Peptide (CSP)-mediated QS [7]. This stimulates the virulence of the pathogen by analysing the density of the microbial community. *Streptococcus mutans* has a capacity to develop several distinctive features to maintain its unhindered survival in the oral cavity.

In order to reduce the rate of disease progression without affecting the oral microbiome or without making any negative regulation in the oral cavity, disrupting or reducing the cellular communication and biofilm formation is the appropriate manner [8].

Recent studies have begun to shed light on the systemic effects of oral pathogens, including *Streptococcus mutans*, suggesting a possible link between oral infections and cardiovascular diseases, diabetes, and other conditions. However, these associations are not fully understood, and the mechanisms through which *Streptococcus mutans* may contribute to such systemic outcomes require further elucidation. Additionally, while various preventive measures against *Streptococcus mutans* colonization and activity have been proposed and implemented, there is a lack of comprehensive reviews that synthesize the evidence on the effectiveness of these strategies.

Moreover, most existing literature tends to focus on isolated aspects of *Streptococcus mutans*' role in oral health, without considering the organism's potential systemic effects or the interplay between different preventive strategies. This fragmented approach limits our ability to develop integrated interventions that can effectively address the multifaceted challenges posed by *Streptococcus mutans*.

This review aims to bridge these gaps by providing a comprehensive synthesis of the current evidence on the virulence factors of *Streptococcus mutans*, its implications for oral and systemic health, and the effectiveness of various preventive strategies. By doing so, it seeks to offer a more holistic understanding of the impact of *Streptococcus mutans* on human health and to identify promising avenues for research, practice, and policy that could mitigate its negative effects.It also highlights the need for future research to explore innovative preventive and therapeutic strategies, investigate the systemic consequences of *Streptococcus mutans* colonization, and develop comprehensive public health interventions aimed at

reducing the prevalence of dental caries and potentially related systemic conditions.

Methods

Research questions

This systematic review aims to address the following research questions:

1. What are the known virulence factors of *Streptococcus mutans*, and how do they contribute to its pathogenicity?
2. What are the systemic implications associated with *Streptococcus mutans* colonization in the oral cavity?
3. What preventive strategies are effective in mitigating the impact of *Streptococcus mutans* on oral and systemic health?

Inclusion and Exclusion criteria

Inclusion criteria:

- Peer-reviewed original research articles and systematic reviews.
- Studies that investigate the virulence factors of *Streptococcus mutans*.
- Research exploring the systemic implications of *Streptococcus mutans* colonization.
- Studies evaluating preventive strategies against *Streptococcus mutans*.
- Articles published in English.
- Studies published within the last 10 years to ensure the relevance and recency of the data.

Exclusion criteria:

- Non-peer-reviewed articles, opinion pieces, and editorials.
- Studies on animals unless they provide insight into human health implications.
- Articles not available in full text.
- Studies not conducted on *Streptococcus mutans*.
- Articles published in languages other than English.

Search strategy

A comprehensive literature search was conducted using the following electronic databases: PubMed, Scopus, Web of Science, and Google Scholar. The search strategy was designed to include a combination of keywords and Medical Subject Headings (MeSH) terms related to "*Streptococcus mutans*," "virulence factors," "dental caries," "oral health," "systemic health," and "preventive strategies." Boolean operators (AND, OR) were used to combine search terms. An example search string for PubMed might be: ("*Streptococcus mutans*" [MeSH]) AND ("virulence factors" OR "pathogenicity") AND ("dental caries" OR "oral health") AND ("systemic health" OR "systemic implications") AND ("preventive strategies" OR "prevention"). The search was limited to studies published from January 1, 2014, to the present to focus on recent evidence. Reference lists of included studies and relevant reviews were also scanned to identify additional studies not captured by the database searches.

Study selection

The study selection process involved two phases. In the first phase, titles and abstracts were screened independently by two reviewers for relevance based on the inclusion and exclusion criteria. Discrepancies between reviewers were resolved through discussion or consultation with a third reviewer. In the second phase, full texts of potentially relevant studies were retrieved and independently assessed for eligibility by the two reviewers. Reasons for exclusion at this stage were documented.

Data extraction

Data from included studies were extracted using a standardized form developed for this review. Extracted information included study characteristics (author, year of publication, country), study design, sample size, participant characteristics, details of the interventions (if applicable), outcomes measured, key findings related to the research questions, and conclusions. Data extraction was performed independently by two reviewers, with discrepancies resolved through discussion or by involving a third reviewer.

Quality assessment

The quality of included studies was assessed using appropriate tools based on the study design. For example, the Newcastle-Ottawa Scale was used for cohort and case-control studies, while the Cochrane Collaboration’s tool for assessing the risk of bias was applied to randomized controlled trials. Each study was independently assessed by two reviewers, and discrepancies were resolved through discussion.

Data Synthesis

Data were synthesized narratively to address each of the research questions. Due to the expected heterogeneity in study designs, interventions, and outcomes, a meta-analysis was deemed not feasible. Instead, findings were grouped by theme (e.g., virulence factors, systemic implications, preventive strategies) and summarized, highlighting patterns, trends, and inconsistencies across studies.

Putative virulence factors of *Streptococcus mutans*

An oral cavity contains distinct habitat for the adherence of microorganism and becomes colonized as microbial community. Microorganisms from the oral cavity have been shown to cause a number of oral infectious diseases, including caries (tooth decay), periodontitis (gum disease), endodontic (root canal) infections, alveolar osteitis (dry socket), and tonsillitis. *Streptococcus mutans*, which is most primarily responsible to be the causative agent of dental caries in humans. Its presence in the oral cavity endorses the development of oral infections and other systemic diseases that are fatal [9].

When discussing about the most sustainable disease that cause the cariogenic infection by the factors associate with adhesion, acidogenicity and tolerance towards the acids **(Figure 1)** [10]. These factors coordinate together and cause dental plaque by altering the ecology of the oral cavity. Then it is coupled with the major factor which is the production of enormous number of extracellular polysaccharides (EPS) or glucans that are produced by the utilization of dietary fermentable carbohydrates (sucrose and fructose) that are catalysed by the enzymes glucosyltransferases (GTFs) and fructosyltransferases (FTFs) respectively. These glucan molecules provide adhesive interaction to *Streptococcus mutans* and also contribute towards structural integrity of dental plaque **(Figure 2)** [11].

The main implication of disease pathogenesis occurs by the microbial cell diversity that are densely populated in the oral cavity. The diversified use of sucrose as a sole carbon source by *Streptococcus mutans* delivers acidic end-products. As the pH falls down by the magnitude of acids that are produced by *Streptococcus mutans*, it greatly increases the cariogenic flora and highly enhance the tooth eruption. These multiple characters together form a complex condition which causes demineralization and progressive destruction of dental enamel [12].

The biofilm regime of *Streptococcus mutans* is enumerated by multifactorial genes that include comABCDE by TCSTS that highly regulates the expression of virulence gene factors. These genes mediate several signalling molecules that are responsible for the enhancement of genetic competence against the host immune system [13]. Development of biofilm protects the microbial group from various host factors and also provides the property of resistance that postulate them as a resistant strain. The emergence of multi resistant strains has been reduced by the development of various anti-virulent drugs such that its count of spreading the disease can be controlled. So it has been pointed that the cell signalling mechanism emerges as an intense component of the biofilm construction.

Different forms of surface adhesion molecules secreted by *Streptococcus mutans*

Adherence is considered as an initial attachment of *Streptococcus mutans* in the dental enamel and causes the intrusion of caries by using ample number of genes [14]. The genes which are responsible for the inducement of virulence are associated with the intercellular communication system, specific regulators for carbohydrate metabolism, environmental sensing systems, and adhesion promoting genes, etc.

And there are different kinds of salivary proteins as proline rich proteins (PRPs), cystatins, histatins, proline-rich mucins (mucin-rich proteins), lactoferrin, lysozyme, amylase, albumin, IgM and IgG that highly interact with *Streptococcus mutans* depending on the time of pellicle formation [15]. GTFs, they synthesize exopolysaccharide glucan molecules for which specific differential receptors are produced by GBPs. The presence of glucan binding domain on GTFs that acts as a receptor for glucans. So the aggregation of *S. mutant* cells is due to the initial development of GTFs and GBPs. In the differential mechanism of *Streptococcus mutans*, they adhere via sucrose dependent manner which are mediated by extracellular enzymes [GTFs, FTFs and glucan binding proteins (GBPs)], these play a well-established role in the virulence of the pathogen. In other way, sucrose-independent mechanisms can also foster microbial colonization by providing binding sites for bacteria [16].

Tolerance mechanism as aciduricity of *Streptococcus mutans*

Streptococcus mutans has a great potential towards acidic environment thus it influences the other microorganism by lowering the pH in the presence of fermentable carbohydrates. When the amount of sugar level is increased in the diet, it favours the plaque causing microorganisms and alters an ecological shift in the actual habitat. As the salivary glycoproteins already provide sufficient nutrition to the existing microorganism and also comfort them for their survival, in addition the external sugar source through diet may further enhance the probability of the virulence mechanism [17].

The uptake of sugar molecules have been transported across the membrane by the system called trehalose-specific phosphotransferase (PTS) or sucrose-specific PTS. This system mainly functions by catalysing the phosphorylation of entering sugar molecules and translocates across the cellular membrane, so these sucrose substrates have been converted into sucrose-6-phosphate. The process of translocation is mainly mediated by the action of permeases that are categorized into three main enzymes as glucose permease, mannose permease and mannitol permease. These enzymes can probably exist as homodimers or oligomers through the phosphorylation of sugar molecules via PTS, they mediate multiple-sugar metabolism systems [18].

The mechanism of phosphoenolpyruvate (PEP): sugar phosphotransferase system (PTS) deals with an energy-coupling proteins as enzyme I, HPr (Heat stable phosphor carrier protein) which is cytoplasmic protein and enzyme complex II or otherwise denoted as sugar-specific permease and they are membrane bound. This is mainly mediated by signal transduction system in which the enzyme I acts as a phosphoryl carrier that transfers to HPr then this phospho-HPr which then phosphorylate to a specific-sugar permease via enzyme II complex [19]. This enzyme II complex is generally a single polypeptide chain comprising of pair of enzymes as II-III or enzyme IIB-IIA. These enzyme complexes were exhibited as hydrophobic transmembrane domain that binds and transports the incoming sugar, and hydrophilic domain that contains the phosphorylation site that catalyses and transports the sucrose substrate [20].

Thus, it is the basic principle behind the transportation and processing of sugar substrate which then fermented and produce acids, the more amount of carbohydrates were fermented by glycolytic pathway contributes to high level of acid generation. In dental caries, the key substrate is considered as sucrose, thus the large amount of sucrose in the diet readily enhances the acid-tolerant bacteria. It is estimated that about ≈90% of the sucrose is taken up by the caries forming *Streptococcus mutans* and ultimately leads to huge acid production. The excess of acid outcome shows severe consequences in the oral cavity especially to the dental enamel, thus the property of aciduricity is considered as a major virulence factor of *Streptococcus mutans* over dental caries [21].

Maintenance of intracellular pH and acid adaptation of *Streptococcus mutans*

Low pH fluctuation is the main cause for the occurrence of dental caries by *Streptococcus mutans* using the continuous exposure of fermentable carbohydrates by the host. These substrates of sugar components were exclusively metabolised by the oral pathogen and produce acid as an end-product. These acid by-products enhance the demineralization of dental enamel and also restrict them from remineralization process [22]. The ability of *Streptococcus mutans* to survive in the low pH is due to the maintenance of transmembrane pH gradient i.e., ΔpH. Proton translocation of F_1F_0-ATPase can upregulate the extrudability of H^+ which then the external environment becomes more acidic that is considered as an important element of *Streptococcus mutans* to survive in the acidic environment [23].

Streptococcus mutans shows rapid response towards acid stress and also creates acidic environment to eradicate the other rival things. And this *Streptococcus mutans* creates microenvironment changes both to the physiological and transcriptional level by disrupting the biological macromolecules that ultimately affects the metabolic pathway [24]. This threat of acid-damage by *Streptococcus mutans* was collectively called as acid-tolerance response (ATR).

The permeability of protons was the main cause for the low pH but the plasma membrane becomes the primary defender against acids by being impermeable to protons. In addition, the cytoplasm remains neutral even though the extracellular environment is acidic. The speciality of *Streptococcus mutans* is that it gives special attention for the maintenance of intracellular pH by one unit of pH higher than the external environment

in order to prevent the acid-sensitive macromolecules [25].

The significance of monounsaturated fatty acids in the plasma membrane helps *Streptococcus mutans* for their survival in the acidic environment. The biosynthesis of fatty acids are mediated by the action of FabA. At low pH, unsaturated fatty acids (UFA) were predominately seen with 18-20 carbon chains, the deficiency in the carbon chains of UFA can cause adverse effects in the ΔpH such as ATPase and hence the ability to cause caries infection will be reduced [26]. UFA have direct impact on the permeability of the protons into the membrane and its related transmembrane proteins in maintaining the stability of ΔpH.

Life on dental surface: **Biofilm of *Streptococcus mutans***

Under culture conditions, *Streptococcus mutans* shows well-defined growth in an anaerobic environment, in case it is subjected to aerobic circumstances, it leads to the reduction of microbial ability to form biofilm. It is processed under signal transduction system, so an availability in oxygen makes alteration on the bacterial cell surface and modifications of autolysins, as increased expression of these autolysins inhibit the formation of biofilm [27]. Biofilm forms a matrix-like structure and provides shelter for the microorganism that are stable to the precise environment. Microbial cell to cell adhesion in the oral environment and their impact on bacterial adhesion and biofilm formation have begun to be recognised [28].

Analysis of the biofilm potential of *Streptococcus mutans* contributes to the process between protein-bacterium interactions. A single cell cannot make a biofilm rather it is suspended in the extracellular substance which creates them to form clusters and become as inseparably associated biofilms [29]. Biofilm patterns are exemplified as a three dimensional structure by their exopolysaccharide matrix format that helps them protecting the microorganism from external factors. The pathogenicity of the microbe is highly determined by the range of damage caused by the microorganism itself and the reaction of immune regulation in response to a pathogen. Thus, the infection course and cure is determined by the host's immunological defence against the pathogen [30].

The process of biofilm development starts with the salivary components that are mediated by the protein molecules as amylase, lysozyme, mucin, histidine, proline-rich proteins, lactoferrin, peroxidase and some of the bacterial components as GTFs, FTFs and lipoteichoic acid ,etc. [31]. These selective salivary proteins are absorbed onto the tooth surface thus forming a protein biofilm called acquired enamel pellicle and also the salivary system of host is filled with vast variety of nutrients that support the pre-existing and invaded microorganism for their survival so this forms a basis for the colonization of microorganism in the oral cavity and leads to the aggregation of *Streptococcus mutans* and the related organism with the pellicle as shown in **Figure 3**.

Mechanism of action by cellular communication

The potential target for *Streptococcus mutans* is to adhere onto the tooth surface that enhances the cariogenic intervention. *Streptococcus mutans* has evolved to depend on a biofilm lifestyle for survival and persistence in the oral cavity. Combined with its role as an opportunistic pathogen, it has become the best-studied example of a biofilm-forming, disease-causing *Streptococcus* as shown in **Figure 4** [32].

The formation of dental plaques is mainly carried out by stepwise manner as:

The oral cavity naturally contains the salivary molecules that are rapidly adsorbed by the enamel as soon as tooth has been cleaned. So, the enamel is coated with the complex mixture of components that include glycoproteins, acidic proline-rich proteins, mucins, bacterial cell debris, exoproducts, and sialic acid [33].

The subsequent interaction of bacteria with this acquired pellicle through several specific cell to surface interactions. However, the attachment of pathogenic microorganism needs primary colonizers for the peripheral adherence over the tooth. The primary colonizers were easily influenced by the environmental parameters such as osmolarity, carbon source and pH [34].

And the *Streptococcus mutans* attach to the primary colonizers via cellular interactions by binding with their specific GBPs. Likewise, the bacteria extend their growth appearance and construct the layers of biofilm on the teeth that are build up further and harden to cause severe dental plaques which harsh the teeth along with the gumline [35].

Extracellular (ComABCDE) and intracellular (ComRS) pathway system in *Streptococcus mutans*

The regulation of CSP signalling system of QS is considered as a potential virulence factor for the induction of genetic competence, this CSP-pheromone molecules are sensed outside the cell. The expression of gene products is encoded by two genetic loci which are *comAB* and *comCDE*. This is a series of genes that forms an operon in which *comAB* which encodes for ATP-binding cassette transporter (*comA*) and an accessory protein (*comB*) to *comA* and this ABC transporter is considered as a pre-processing mediated reaction [36]. The secretory protein encoded by *sepM* produces an enzyme protease that is highly involved in the processing and export of these CSP molecules for the final or post processing reaction. Another loci is *comCDE* in which *comC* encodes for the precursor to the CSP, histidine kinase is encoded by *comD*, and *comE* encodes for response regulator. In this, the product of *comD* acts as a receptor for processed CSP and the response regulator that activates the operon *comAB* and *comCDE*. There is a threshold concentration for CSP, when it reaches that point, it starts to bind with their respective receptor [37].

This *comE* further regulates the transcription of some of the genes as bacteriocin related genes in a direct way, and indirectly regulating sigma factor. *CipB* and *cipL* are the genes that activates *comRS* mediated QS mechanism [38]. This *comRS* QS system is mainly composed of a double-tryptophan containing signal peptide XIP that is encoded by *comS* and their transcriptional regulator is *comR*. The *comRS* regulation system is mainly sensed inside the cell after its internalization via an *Opp* transporter in peptide-free medium because it directly controls the expression of *comE*. The complex formation of XIP/*comR* activates further transcription of *comR* and *comS* genes which creates positive feedback, and induces the expression of *sigX* which is mainly involved in the control of competence regulon as shown in **Figure 5**.

Preventive strategies against *Streptococcus mutans*

Enhanced oral hygiene practices

Regular and effective oral hygiene practices are the cornerstone of preventing dental caries caused by *Streptococcus mutans*. Brushing twice a day with fluoridated toothpaste and daily flossing can significantly reduce the bacterial load in the oral cavity. Fluoride works by enhancing remineralization of the tooth enamel and inhibiting the cariogenic metabolic activity of *Streptococcus mutans*. Additionally, mouthwashes containing antimicrobial agents such as chlorhexidine can offer a supplementary method to control bacterial populations when used judiciously.

Dietary modifications

Diet plays a pivotal role in the proliferation of *Streptococcus mutans*. The bacterium metabolizes fermentable carbohydrates, predominantly sugars, to produce acids that demineralize tooth enamel. Limiting the intake of sugary foods and beverages, coupled with the consumption of a balanced diet rich in fiber, vitamins, and minerals, can diminish the risk of caries. Encouraging the consumption of sugar substitutes such as xylitol, which *Streptococcus mutans* cannot metabolize to produce acid, offers an additional preventive strategy.

Use of probiotics

The introduction of beneficial bacteria to the oral microbiome, known as oral probiotics, has emerged as a novel approach to counteract *Streptococcus mutans*. Specific strains of Lactobacillus and Bifidobacterium have demonstrated the ability to inhibit the growth of *Streptococcus mutans* by competing for adhesion sites, producing antimicrobial substances, and modulating the host immune response. Regular consumption of probiotic-containing products may therefore contribute to a balanced oral microbiome and reduced caries risk.

Innovative dental materials

Recent advancements in dental materials have focused on the development of products that not only restore the tooth structure but also prevent future caries. Bioactive glass used in dental fillings releases ions that neutralize the acidic environment, inhibiting *Streptococcus mutans* activity. Similarly, dental composites and sealants incorporating quaternary ammonium compounds exhibit potent antimicrobial properties against *Streptococcus mutans*, preventing biofilm formation on the surfaces of teeth.

Vaccination efforts

Although still in experimental stages, research into vaccines targeting *Streptococcus mutans* holds promise for a long-term solution to prevent dental caries. By inducing an immune response against specific antigens of *Streptococcus*

mutans, such vaccines aim to reduce colonization and virulence of the bacterium. While challenges remain, including ensuring targeted specificity and safety, early preclinical trials show potential for future development.

Public health initiatives

Public health measures, including community water fluoridation and educational programs on oral health, play a crucial role in reducing the prevalence of dental caries at the population level. Water fluoridation has been proven to decrease the incidence of caries significantly by providing continuous exposure to fluoride. Educational initiatives that promote awareness of oral hygiene practices, dietary habits, and regular dental check-ups are essential for empowering individuals to take proactive steps in preventing dental caries.

Figure 1. Strategical development of caries on dental surface by *S. mutans.*

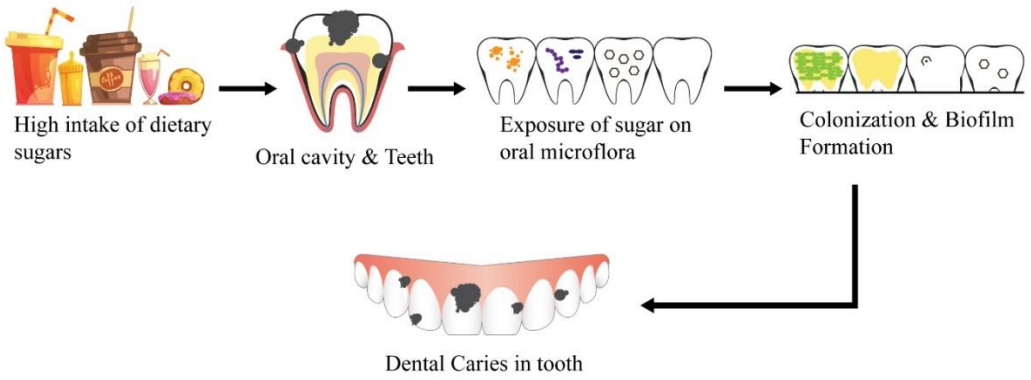


Figure 2. Enhanced biofilm formation by adhesion to pre-formed glucans and P1 proteins on the tooth surface.

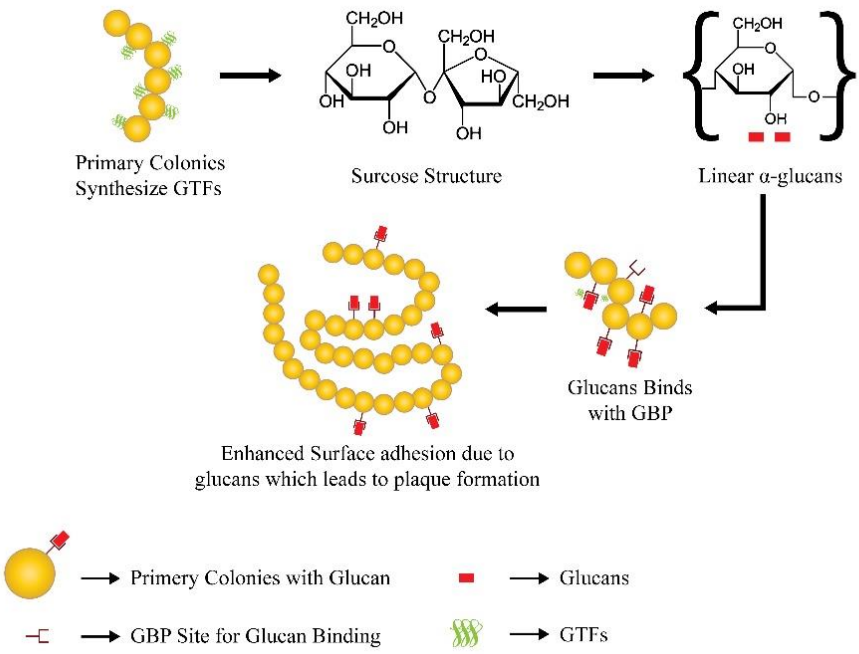


Figure 3. Development of dental plaque.

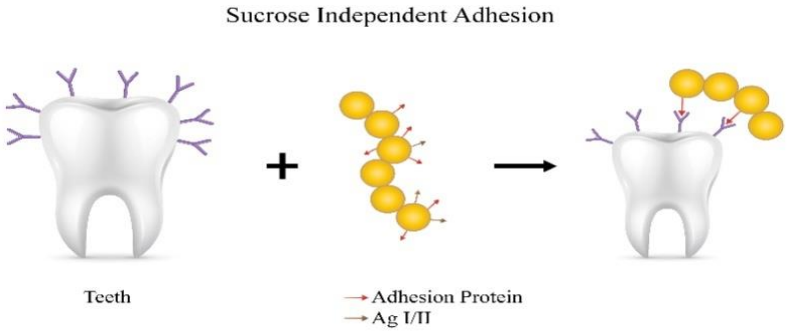
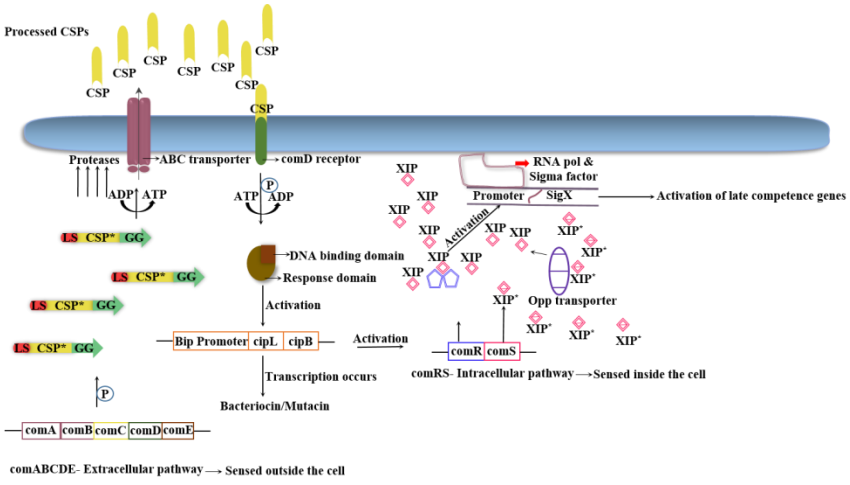


Figure 4. Cellular construction on dental surfaces.

Figure 5. Architecture of *S. mutans* quorum sensing (QS) mechanism.



Conclusion

This review concludes that the prevention of caries is necessary in the early phase of dentate period. The outcome of disease progression is complicated through a sequential reaction performed by the pathogen to place their survival in the human system. With the initiation of demineralization till the deposition in the heart valves, the pathogen arrests and blocks the physiological activities. Oral health practices, hygienic behaviour and diet habits are the potential factors to cease the growth of cariogenic bacteria. Numerous caries management strategies are evolved but the actual preventive agent should halt the caries progression and appears to prevent the development of new caries.

The review also highlights the importance of preventive strategies in managing the risk posed by *Streptococcus mutans*, including improved oral hygiene practices, the use of antimicrobial agents, the application of dental sealants, and dietary modifications. Despite the availability of various interventions, the challenge remains in implementing these strategies effectively at the population level and in individual patient care.

Recommendations

For practice

1. Dental professionals should emphasize the importance of regular oral hygiene practices, including proper brushing and flossing, to reduce the colonization of *Streptococcus mutans*.
2. The use of fluoride toothpaste and mouth rinses should be encouraged as part of routine oral care to inhibit the growth of *Streptococcus mutans* and enhance the remineralization of tooth enamel.
3. Dietary counseling should form an integral part of dental care, focusing on reducing the intake of sugary and acidic foods and beverages that can promote the growth of *Streptococcus mutans.*

For policy

1. Public health policies should support the integration of oral health education into general health promotion programs to raise awareness of the importance of controlling *Streptococcus mutans* colonization.
2. Policies aimed at reducing the consumption of sugary snacks and beverages through taxation, labelling, and restrictions on marketing to children can contribute to reducing the prevalence of dental caries.
3. Access to preventive dental care, including fluoride varnish applications and dental sealants, should be expanded, especially in underserved communities.

For future research

1. Further studies are needed to elucidate the mechanisms underlying the systemic effects of *Streptococcus mutans* colonization, particularly its potential contributions to cardiovascular diseases and diabetes.
2. Research into the development of novel antimicrobial agents and vaccines targeting *Streptococcus mutans* is warranted to provide additional tools for caries prevention.
3. Longitudinal studies assessing the effectiveness of integrated oral health interventions on reducing *Streptococcus mutans* colonization and preventing dental caries in diverse populations would be valuable.

Conflicts of intereset: None to be declared.

Financial disclosure: None.

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