



# From Plaque to Pulse: Exploring the Unexpected Impact of Streptococcus Mutans on Oral and Heart Health

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## **Authors' contributions**

*This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.*

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## **ABSTRACT**

Oral health is often overlooked as a key factor in overall well-being, yet emerging research suggests a strong link between dental bacteria and cardiovascular diseases. Among these bacteria, *Streptococcus mutans*—a well-known contributor to tooth decay—has been increasingly recognized for its potential role in heart health. This review explores the mechanisms through which this bacterium moves beyond the oral cavity, potentially influencing systemic inflammation, arterial plaque formation, and vascular diseases linked to heart conditions. While *S. mutans* primarily causes dental caries through its production of biofilms and acidification of the oral environment, its

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ability to invade the bloodstream through gum infections raises concerns about its broader health impact. Studies indicate that oral bacteria, when introduced into circulation, can contribute to vascular inflammation, increasing the risk of atherosclerosis and other cardiac complications. The bacterium's virulence factors, including adhesins and extracellular polysaccharides, facilitate its survival in different tissues, potentially exacerbating heart disease. By reviewing recent findings, this article aims to bridge the gap between dentistry and cardiology, highlighting the need for interdisciplinary collaboration in healthcare. Understanding the connection between oral and cardiovascular health underscores the importance of proactive dental care, encouraging better hygiene practices as a preventive strategy for systemic diseases. Moreover, this discussion raises awareness about the necessity of monitoring oral infections as a potential contributor to heart disease, emphasizing a holistic approach to patient health. Ultimately, taking care of one's teeth isn't just about preventing cavities—it might also be a crucial factor in maintaining cardiovascular health. A deeper appreciation of this connection could pave the way for more comprehensive medical strategies, ensuring that oral health is integrated into broader disease prevention frameworks.

**Keywords:** Oral health; streptococcus mutans; dental plaque; cavities; heart health; cardiovascular disease; inflammation; microbial translocation.

## 1. INTRODUCTION

Heart health and dental health have long been interlinked. It has been thoroughly examined and brought up how dental hygiene influences the onset of heart disease. Most professionals in the area agree that poor dental hygiene can increase the risk of infective endocarditis in vulnerable people (e.g., individuals with valve damage from acute rheumatic fever). Patients currently have a much longer life expectancy and significantly superior surgical outcomes owing to the increasingly advanced treatment of valvular heart disease (Mahajan et al., 2025). However, these beneficial outcomes are marked by a greater chance of getting infective endocarditis and its complications. Bacterial endocarditis has a significant risk factor related with poor oral health, particularly periodontal condition. There is a positive correlation between gingival inflammation and the rate and severity of bacteremia (Lowry et al., 2005). A dangerous bacterial biofilm that may accumulate on tooth surfaces is considered to be the cause of dental caries, an infectious illness. Acid-tolerant bacteria proliferate and change physically to an elevated acidic environment once the microbial composition changes, producing a dysbiotic shift in the biofilm. When the proportion of some bacteria shifts, a commensal bacterial community ultimately develops into a highly pathogenic one. This occurrence is known as dysbiosis. Tooth bleaching, or caries, is the result of this change, which eventually raises the biofilm's acidic potential (Bumm & Folwaczny, 2021). Dental caries involves interactions between the tooth structure, the microbial biofilm

formed on the tooth surface, and sugars, as well as salivary and genetic influences. The dynamic caries process consists of rapidly alternating periods of tooth demineralization and remineralization, which, if net demineralization occurs over sufficient time, results in the initiation of specific caries lesions at certain anatomical predilection sites on the teeth. It is important to balance the pathological and protective factors that influence the initiation and progression of dental caries (Daboor et al., 2015).

Protective factors promote remineralization and lesion arrest, whereas pathological factors shift the balance in the direction of dental caries and disease progression. The main reason of the widespread decrease in tooth decay in the last few years, based on many sources, is regular application of fluoride toothpaste (Nakano et al., 2008). These toothpastes work by balancing the oral biofilm in favor of health. There is no correlation between pain and discomfort and the severity of a caries lesion. Serious systemic consequences, such as tooth loss, treatment-related death (due to anesthesia), and the spread of local infections, may occur from infection and sepsis caused by caries that spreads to the dental pulp. On contrary, a severe toothache can be disruptive when it occurs (Pitts et al., 2017). Patients who required an antibiotic endocarditis prophylaxis (AEP) had a higher number of lost and filled teeth due to caries, as well as a higher Decayed, Missing, and Filled Teeth (DMFT) index. This may have resulted in more frequent dental procedures since these high-risk patients were more likely to understand the value of maintaining good oral health.

However, there was no discernible difference in the quantity of untreated decaying teeth. As a result, it is unclear whether the variation in the DMFT index was brought about by higher caries prevalence in high-risk patients or more frequent dental procedures (Hollatz et al., 2019). A multimodal preventative strategy to potentially fatal diseases is required as more patients with coronary heart disease (CHD) reach adulthood as a result of advancements in medical and surgical care. Optimizing illness awareness is crucial to promoting patient self-management (Hollatz et al., 2019). Despite the fact that the oral cavities of a wide range of mammalian species are home to billions of microbial cells, the majority of scientists identified that *Streptococcus mutans* is the microorganisms that causes dental caries, or decay. Over the years, scientists have studied the specific role of *S. mutans* and what allows it to survive when non-pathogenic bacteria cannot. This review will seek to deal with this specific question. Furthermore, it will focus on the ecology of the oral cavity and seek to confirm a concept known as the "ecological plaque hypothesis" through a number of probes. The role of *S. mutans* will be suitably explained by this theory, which will also pave the way for effective preventative measures (Friedman, 2011). Dental caries is caused by the extracellular polysaccharide that *S. mutans* generates from sucrose. This extracellular material has a (1-3) glucose linkage that facilitates the bacterium's adhesion. This polymer also seeks to provide energy when there is a deficiency of any essential carbohydrates. Lipo

Teichoic Acid, which *S. mutans* also produces, entirely binds to the outer enamel and enhances colonization. It adheres superficially to the tooth, breaks down sugar for energy, lowers pH, and creates an acidic environment, which demineralizes the tooth's outer layers, including the enamel and dentine. Rarely, if no previous medication is taken, the process progresses and eventually results in dental caries. Consequently, *S. mutans* is not only the primary bacterium responsible for plaque formation but also for the initiation of dental caries (Mozos & Stoian, 2019).

### 1.1 Importance of Oral Systemic Health Connections

The idea that dental health may have a big impact on occurrences in other parts of the body is not new, but it has changed throughout time. 2–5 Miller's 1891 article, "The Human Mouth as a Focus of Infection," is a well-regarded early work. Six Being involved in Robert Koch's lab, whose postulates were used to establish the microbiological etiologies of infectious illnesses, given Miller a keen understanding of the role bacteria play in disease etiology. Miller suggested that a number of systemic infectious illnesses, brain abscesses, lung disorders, stomach issues, and other conditions that occur in areas outside than the mouth cavity might be caused by oral microbes or their byproducts (Barnett, 2006). It has been maintained that the oral cavity is "the window to general health." It is now common to say things like "The mouth is part of the body" and "You cannot have good

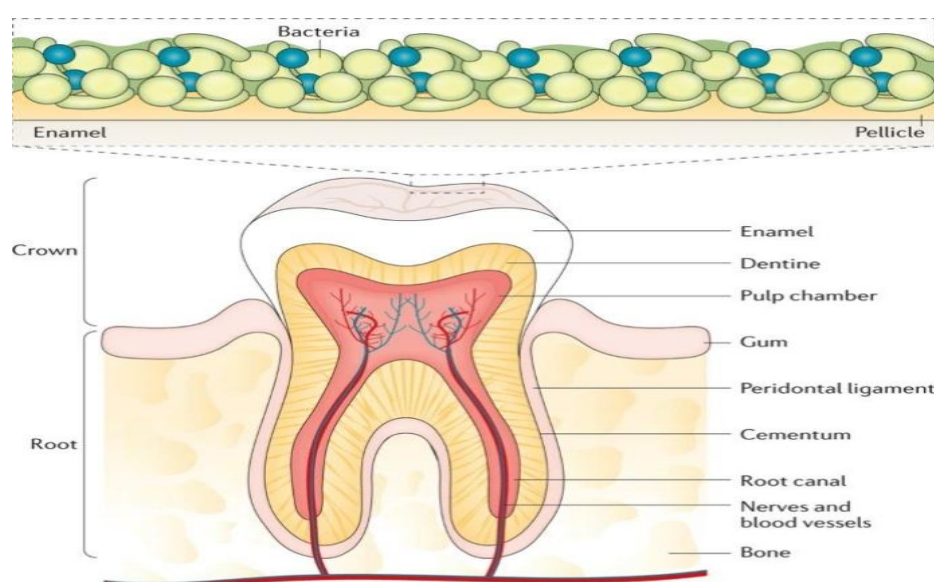


Fig. 1. Structure of Tooth with bacterial infection

general health without good oral health," according to Seymour. The oral cavity also serves as the meeting point for dental and medicine, two relatively independent fields with a shared objective of enhancing patients' health and quality of life. Each profession is based on the fundamental idea that patients' health, welfare, and quality of life will be improved overall by suitable interventions within the discipline's framework.

## 1.2 Oral Cavity

The oral cavity and mouth serve as important spots for the body's interactions with the outside world. The oral cavity is involved in several essential physiological processes, including speech, chewing, swallowing, and the initial phases of digestion. Also, the mouth supports psychological identity. The oral cavity has several surface types, and each is host to a distinct group of 500–700 different species of bacteria, viruses, fungi, and protozoa, many of which have not been developed and many of which are extremely virulent. The composition of the oral microbiome is greatly influenced by the degree of dental cleanliness. Gram-positive cocci and rods, as well as some gram-negative cocci, predominate in the simple flora of people who practice good oral hygiene, whereas anaerobic gram-negative organisms predominate in the more varied and complicated flora of people who maintain poor oral hygiene. Saliva and gingival crevicular fluid make up the majority of the complex fluid combo that surrounds the oral cavity and is crucial to maintaining a healthy oral environment. Like many other body functions, saliva has an ideal range; modifications to its chemical composition, underproduction (xerostomia), and overproduction (sialorrhea) can all have an effect on health in every direction. Saliva and bacteria directly lead to the formation of the dental biofilm, which most of the population refers to as plaque. Nowadays, it has become more recognized that not all plaque is harmful and that the interaction between bacteria and saliva helps shield teeth by minimizing exposure to food acid and preserving mineralized. When salivary molecules are absorbed on the surface of the tooth, the first bacteria cling to them. As they proliferate, the bacteria create a matrix of polymers to which further bacteria can adhere. The biofilm that is connected to healthy teeth and gingiva is characterized by a commensal microbiota of streptococcal species. By preventing the colonization of more pathogenic species, these

organisms show themselves to be useful (Kane, 2017).

## 2. SYSEMIC CONDITIONS IMPACTED BY ORAL HEALTH

Economic, social, psychological, and physical health are among the well-researched advantages of maintaining good dental health. The quality of life decreases with the number of lost teeth, according to Garcia et al. Additionally, poor dental health might affect dietary intake and cause problems with chewing. These connections are clear, but greater collaboration is needed in the areas of the major effect and association between severe systemic illnesses and oral health issues. Both the health of people and the overall healthcare system are severely impacted by chronic health disorders. Regular dental treatment helps identify people who are at high risk for more serious systemic illnesses in while also preventing periodontal disease. The following are some of the systemic conditions that are affected by oral conditions, either directly or indirectly: diabetes, atherosclerotic disease, pulmonary illness, pregnancy, birth weight, osteoporosis, and renal disease (Kane, 2017).

### 2.1 Dental Caries and Streptococcus Mutans

J. Clarke termed the bacterium he acquired from carious lesions in 1924 *Streptococcus mutans* because he assumed the oval-shaped cells he noticed were mutant streptococci. However, *S. mutans* only became widely recognized in the scientific community in the late 1950s. By the mid-1960s, however, clinical and animal-based studies conducted in laboratories showed that *S. mutans* was a significant causative agent in dental caries. *S. mutans*'s natural environment is the human mouth, more especially the dental plaque, a multispecies biofilm that forms on tooth surfaces. Most people agree that *S. mutans*'s cariogenic potential stems from three essential characteristics: (i) the capacity to produce large amounts of glucan extracellular polymers from sucrose, which promote a constant colonization of hard surfaces and the formation of the extracellular polymeric matrix in situ; (ii) the capacity to transport and transform a number of carbohydrates into organic acids (acidogenicity); and (iii) the capacity to survive in stressful environmental conditions, especially low pH (aciduricity). Although *S. mutans* does not cause dental caries on its own, research from multiple labs has clearly shown that it can change the

local environment by forming a low-pH, extracellular polysaccharide (EPS)-rich milieu, providing other acidogenic and aciduric species a favorable niche to grow in (Lemos et al., 2019). The surface protein antigens of *Streptococcus mutans*, a Gram-positive capable anaerobic bacterium that has become recognized as a dental caries pathogen, have been characterized in order to clarify their function as virulence factors. Three forms of GTFs (GTFB/GTFC/GTFD) have been identified to be involved in sucrose-dependent adhesion to tooth surfaces. Glucosyltransferases (GTFs), protein antigen (PA), and glucan-binding proteins (Gbps) have been determined to be important surface protein antigens of *S. mutans*. According to this, GTFB and GTFD convert sucrose into insoluble and soluble glucans, accordingly, whereas GTFC contributes to the synthesis of both glucan types. However, it has been demonstrated that PA, which also included SpaP, antigen I/II, antigen B, SR, IF, P1, and MSL-1, is connected with sucrose-independent initial adhesion to tooth surface. The glucan-binding characteristics of *S. mutans*'s four Gbps forms (GbpA, GbpB, GbpC, and GbpD) make them virulence factors for dental caries. By inactivating the genes encoding each protein antigen, isogenic mutants defective of target proteins have been created for use in in vitro analyses with the goal of defining the mechanism of dental caries. The major involvement of these protein antigens with dental caries was demonstrated in animal tests including particular pathogen-free rats orally infected with isogenic mutants and their parent strain (Nakano et al., 2008). The mouth, pharynx, and intestine are the main places where *S. mutans* streptococci exist. The microbe is non-motile, catalase negative, and has four variants, which are named c, e, f, and k based on the chemical makeup of the serotype-specific rhamnose-glucose polymers. Attachment to the enamel surface, the generation of acidic metabolites, the ability to form glycogen reserves, and the capacity to produce extracellular polysaccharides are some of the main elements of dental caries, which are caused when any changes occur in the oral cavity that occur during increased glucose consumption, modifying the ecosystem's homeostasis to notably acidophilic bacteria that are frequently involved in tooth loss., Mutans streptococci are vigorous acid producers that create an acidic environment, which makes them toxic for the development of teeth. One prevalent chronic condition in children is dental caries. In fact, it can often be severe and manifest in young

children just after the emergence of their first teeth. Early childhood caries hurts nearly every child and degrades their health, but for a small percentage of kids, it can lead to serious illness, hospitalization, and in rare cases even death. Consequently, dental caries is the primary cause of tooth loss in both young adults and children. It has been claimed that Dental caries can alternatively be described as a contagious bacterial illness affecting dental teeth that results in minimal breakdown and loss of hard structures. It is caused by infectious bacteria and easily accessible carbohydrates in food and drink. It comes from a Latin word that means "rot or decay." *S. mutans* is a common tenant of dental plaque and has been identified as the primary cause of cavities. Dental caries is a major cause of stress and negative situations in both children and adults, as it results in pain, discomfort, and costly medical care. These streptococci bacteria are special in that they may colonize tooth surfaces and, under certain conditions, form massive cariogenic biofilms. and create more biofilms with different bacteria and streptococci as well as other organisms. A mutual bacterial association that sticks to a solid (such an intravenous catheter or a denture prosthesis) or to one another while being shielded by an extracellular polysaccharide matrix is called a biofilm. Microbial biofilms are thought to be connected to around 65% of human illnesses. Dental plaque generated on tooth surfaces is a common example of a biofilm. *S. mutans* can also enter the bloodstream, causing temporary bacteremia during stressful conditions such as dental surgeries, oral diseases, dental hygiene, or eating. After entering the bloodstream, this bacterium seeks to cause infectious endocarditis. One useful application for liquid chlorhexidine gluconate is as an antiplaque agent (Daboor et al., 2015).

## 2.2 Dental Decay

Think of a dental cavity as the result of an ongoing tug-of-war between your teeth and harmful bacteria. Every time we eat foods rich in sugar or carbohydrates; we unknowingly fuel the bacteria lurking in our mouths. These bacteria thrive on the remnants of our meals, forming a sticky, invisible film called plaque that clings to the surface of our teeth. The real trouble begins when plaque comes into contact with sugary foods. The bacteria process these carbohydrates for energy, producing acid as a byproduct. This acid slowly dissolves the enamel—the hard, protective outer layer of the tooth—weakening its

structure. Over time, this erosion deepens, leading to tiny holes or cavities. At first, a cavity may not seem like a big deal, but if left untreated, it can penetrate deeper into the tooth, affecting the softer layers inside and even reaching the nerves. This can lead to sensitivity, pain, and, in severe cases, infections or abscesses. But here's the good news—cavities are largely preventable! Regular brushing, flossing, and mindful eating habits can keep plaque in check and minimize the damage bacteria can do. Your teeth work hard every day, so showing them a little care can keep them strong for years to come.

For around twenty minutes, the acid lowers the pH in the mouth and causes the surface of the teeth to lose minerals. The initial sugar exposure triggers this attack, which persists for 20 minutes following the final dose. Minerals in the oral cavity are redeposited on the tooth surface by a dynamic process after the pH is balanced (following acid assaults). While these minerals are being replenished, the tooth surface stays intact. However, enamel breaking down or a "cavity" is ultimately caused by recurrent acid assaults, a protracted pH decrease, and a significant net loss of minerals.

Tooth decay isn't just a sudden event—it's a slow, sneaky process that takes place over time, often without us realizing it. Our teeth are constantly interacting with the foods we eat, especially sugars and starches. When we indulge in something sweet, the bacteria in our mouth feast on it, producing acids as a byproduct. These acids slowly eat away at the protective enamel of our teeth, making them weaker and more vulnerable. Plaque, a sticky film of bacteria, builds up on the surface of our teeth if not cleaned properly. It traps these harmful acids against the enamel, speeding up the decay process. Over time, tiny holes, known as cavities, can form, leading to pain, sensitivity, and, if left unchecked, even infections or tooth loss. It's a battle between care and neglect—regular brushing, flossing, and mindful eating can help protect our teeth from this deterioration. But when we skip these steps, we give decay the perfect conditions to thrive. Our teeth work hard for us every day—it's up to us to return the favor.

## 2.3 Teeth-decay

The cavity may get larger and penetrate deeper into the dentine and pulp if the decay continues and the cavity not recovered. The jaw bone and

other areas of the face and body may get infectious after the pulp is affected.

## 2.4 Bacteria and Dental Plaque

Bacterium and Dental Plaque Science suggests that dental caries is influenced by the bacterium mutans streptococci (MS). The variables related with MS colonization in children include frequent sugar intake, frequent snacking, drinking sweetened beverages before bed, sharing meals with adults, and high levels of maternal MS." The transfer of these germs to the infant can be significantly affected by high levels of MS in the mother's mouth (New Zealand Dental Association, 2008).

## 2.5 The Oral-heart Connection

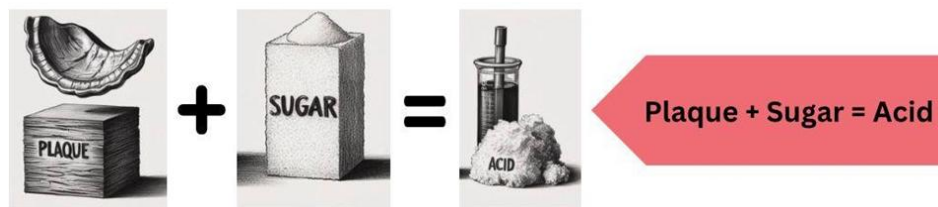
There is an increasing concentration on the association between oral health and overall health. Due to the increasing prevalence of diseases like cardiovascular disease, researchers and dentists have put forward that certain patients' disease The inception may be directly caused by oral germs entering the blood stream. cardiovascular disease will be described along with the bacteria's suspected mode of action. Tooth decay continues to be one of the most significant and most prevalent problems with children's and adults' oral health. Worldwide collaborated and Streptococcus mutans has been identified as the primary cause of dental caries formation. Maintaining the teeth's normal function and condition is essential due to their effect essentially, mastication, and the general shape and appearance of the face. Therefore, a lot of focus is on ways to prevent the accumulation of bacteria in the mouth, which combines with the adverse circumstances to cause cavities. The bacterium is responsible for dental cavities and is also believed and participated to cardiovascular disease. Streptococcus mutans traces have been identified in the bloodstream of individuals suffering from endocarditis, a type of cardiovascular illness, according to a flurry of data (Burks, 2014).

## 2.6 Association between S. Mutans and Cardiovascular Disease

An estimated 100 trillion live cells make up a healthy human person.90% of these cells, however, are just microorganisms that reside in places like the mouth, skin, and digestive system. Actually, each environment has a unique

microorganism composition that, once established in a healthy human, stays constant. Microbial homeostasis is the term for this. The presence of important ecological elements is what keeps the microbial community stable in each unique habitat. These elements consist of oxygen levels, attachment receptors, pH levels, and nutrition. Disease may arise as a result of environmental changes that significantly disrupt the homeostatic balance required to support the microbial species. Almost half of these microorganisms, or around six billion microbial organisms, are found in the mouth cavity alone out of all the regions that have this massive number of microbial cells. The mouth cavity has numerous characteristics that make it a perfect home for microorganisms. The constant flow of saliva is one of these characteristics. Saliva significantly improves the mouth cavity's ecology. Saliva's ionic composition acts as a buffer and repairs enamel, and its pH of 6.95 is perfect for the growth of microorganisms. Saliva also maintains the mouth wet and heated (around 35°C). However, by covering the surfaces of the mouth, saliva may have the most advantageous effect on microbial adhesion: a selective conditioning film in the oral cavity (Friedman, 2011). While initially it was assumed that *Lactobacillus* sp. and *Streptococcus* sp, especially *Streptococcus mutans*, were responsible for the acidification of the biofilm, more recently composed etiological models have shown that many other endogenous bacteria found in the dental biofilm are capable of acidification. Others are able to increase pH values and thus counteract over-acidification.

Dental caries is based on a complex multifactorial etiology, causing interactions between components such as salivary flow and the substance, the structure of dental hard tissues, environmental factors, and genetic susceptibility, even though the formation of a biofilm on the teeth is known to be essential for the initial growth of carious lesions. Carious lesions are largely caused by behavioral factors, including dental hygiene and eating habits. A persistent decrease in pH in the biofilm due to continuous ingestion of fermentable carbohydrates, such as sugar, demineralizes hydroxyapatite and eventually leads in the development of a recognizable caries lesion, involving discoloration and cavitation. As a result, bacteria first penetrate the necrotic tissue before moving on to the periapical tissue, which comprises the surrounding alveolar bone and the apical areas of the periodontal ligament. In these inflammatory lesions, the bacterial flora is typically slightly diverse and complex, consisting of 10–20 different, mostly Gram-negative species. In contrast, if the bacterial infection occurs in teeth that have undergone prior endodontic treatment, the bacterial spectrum is reduced, consisting of 3-6 different gram-positive and gram-negative species, including *Staphylococcus* sp. The described bacterial infection generally triggers an inflammatory process within the periapical tissue, which often has a chronic, asymptomatic course. The lack of clinical symptoms, such as pain or swelling, usually causes the inflammatory process to manifest itself over a longer period of duration, being undetected (Bumm & Folwaczny, 2021).



**Fig. 2. Formation of Acid**  
*Plaque + Sugar = Acid*



**Fig. 3. Tooth decaying**  
*Acid + Tooth = Decay*





**Fig. 4. Model of tooth decay**  
*Plaque + Sugar + Tooth = Decay*

## 2.7 Cardiovascular Disease (CVD)

Heart and blood vessel disorders, including atherosclerosis, coronary heart disease, stroke, and myocardial infarction, are together referred to as cardiovascular disease (CVD). These ailments are caused by a complex combination of environmental and hereditary factors. Chronic infection and inflammation are increasingly being linked to CVD, with biofilm serving as a risk factor. In the literature, the link between oral bacteria and heart disease has long existed. Oral bacteria, particularly *Porphyromonas gingivalis* (periodontitis) and *Streptococcus mutans* (cariogenic), cause platelets to aggregate, which results in the development of thrombus. In individuals with severe periodontal disease, 42% of atheroma's had one or more periodontal infections, according to published research. According to reports, *P. gingivalis* has the ability to aggressively cling to and infiltrate fetal bovine heart and aortic endothelial cells. Furthermore, compared to their oral-healthy peers, individuals with periodontal disease had a 25% increased chance of developing cardiovascular disease, according to 14-year research. The chances of developing cardiovascular disease are 72% higher for men under 50 who have periodontal disease. Furthermore, the risk of strokes, both fatal and non-fatal, was doubled by periodontal disease. It is uncertain if there is a direct or causative link between periodontal disease and CVD, despite the substantial evidence of one. When germs from periodontal disease are released into the bloodstream, they can infiltrate the heart and vascular tissue and cause damage. Individuals who have more oral bacteria generally have bigger carotid arteries, which is a sign of cardiovascular disease. It seems that bacteria

close to infected gingiva cause blood platelets to clump together, which can result in clotting and blockages that can cause heart attacks or strokes. Inflammatory mediators are produced as part of the body's reaction to a periodontal infection; these substances enter the bloodstream and have adverse impacts on the heart and blood vessels. In comparison to control groups, patients with periodontitis have considerably increased levels of inflammatory mediators, including triglycerides and lipoprotein. Increased clotting is linked to an increased risk of heart disease, as is elevated levels of C-reactive protein, a biomarker for inflammation that is linked to periodontitis. The discovery that periodontal disease may be a risk factor for cardiovascular disease is causing oral and medical care to converge. Maintaining good dental health should be essential to preventing heart disease or making pre-existing heart issues worse (Kurtzman et al., 2022).

## 3. ATHEROSCLEROSIS

When fatty patchy deposits develop in the walls of medium or large arteries, a condition known as atherosclerosis occurs, which reduces or stops blood flow. Through its detrimental effects on vascular endothelial cells, which were more noticeable in the damaged endothelium, *S. mutans* accelerated the induction of AS in mice. Endocarditis is infectious. *S. mutans* attaches itself to the exposed collagen in the injured cardiac endothelium, where it is joined by activated platelets through the factor vWF. The *Cbm +/PA*-strain may interact with fibrinogen when it attaches to the Glycoprotein IIb/IIIa receptor on platelets, speeding up the development of redundant organisms and causing IE (Fang et al., 2024).



### 3.1 Mechanisms of *S. Mutans* Entry into Systemic Circulation

*Streptococcus mutans*, the bacteria commonly associated with dental caries, was identified also in the atherosclerotic plaque, suggesting a proatherogenic potential of dental caries, and may also cause bacterial endocarditis. It takes less than 1 minute after an oral intervention for the oral bacteria to reach the heart or the peripheral capillaries. Normally, microorganisms are eliminated within minutes, but in patients with valvular heart disease, bacteremia may cause infective endocarditis (Mozos & Stoian, 2019). A major cause of dental caries, *Streptococcus mutans* (*S. mutans*) has grown to be recognized as a major cause of cardiovascular diseases (CVDs) due to its spreading, virulence factors, and inflammatory potential. It enters the oral cavity by attaching itself to tooth enamel using surface-associated adhesins including glucan-binding proteins (GBPs) and antigen I/II (Agl/II), which promotes the production of biofilms. These biofilms, which are made of extracellular polysaccharides made by glucosyltransferases (Gtfs), serve as a protective matrix that increases the ability of bacteria to antimicrobial drugs and host immunological responses. Dietary carbohydrates are fermented by *S. mutans* causes the creation of lactic acid, which lowers pH and demineralizes enamel, starting the tooth caries process. Its function, however, goes beyond the mouth cavity; it acts as a microbiological link between cardiovascular disease and poor dental health. Transient bacteremia, a condition when *S. mutans* enters the circulation through damaged gingival epithelium, can occur after periodontitis or invasive dental treatments like tooth extractions. Infectious endocarditis develops when the bacteria enter the bloodstream and uses its collagen-binding protein (CBP) to stick to exposed collagen on injured endocardial surfaces, especially heart valves. Serine-rich repeat adhesins (Srr) help bacteria attach to platelets, which encourages platelet aggregation and thrombus development. The formation of a fibrin-rich biofilm-like structure by platelet aggregation protects *S. mutans* from the host immune system, enabling chronic colonization and raising the possibility of embolization, which can result in myocardial infarction or stroke. Through systemic inflammation and immunological regulation, *S. mutans* contributes to cardiovascular disease in addition to its direct vascular colonization. The bacteria trigger

intracellular signaling cascades that lead to the activation of nuclear factor-kappa B (NF- $\kappa$ B) by activating toll-like receptors (TLRs), specially TLR2 and TLR4 on monocytes and endothelial cells. As a result, pro-inflammatory cytokines such interleukin-1 $\beta$  (IL-1 $\beta$ ), interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- $\alpha$ ), and C-reactive protein (CRP) are secreted more often, which leads to atherogenesis, vascular inflammation, and endothelial dysfunction. The development of atherosclerotic plaques is accelerated by chronic inflammation because cytokine-mediated oxidative stress encourages the oxidation of low-density lipoproteins (LDL), which results in the creation of foam cells and arterial constriction. Furthermore, it has been discovered that *S. mutans* invades macrophages, changing their lipid metabolism and promoting the development of foam cells, which is a crucial stage in atherogenesis. Because the bacteria can cause endothelial cells to undergo apoptosis, vascular damage is created worse, compromising blood vessel integrity and encouraging thrombotic events. Molecular mimicry between host proteins and bacterial antigens can also result in autoimmune-like reactions, which exacerbate cardiovascular diseases by having the immune system incorrectly target vascular components. Certain serotypes of *S. mutans*, like serotype k, may be more virulent and have a greater affinity for cardiovascular tissues than other strains, according to recent research. A strain-dependent effect on cardiovascular pathology is suggested by the strain's frequent isolation from valvular heart disease patients. Heat shock proteins (HSPs), which are produced by *S. mutans* under stress, can also function as autoantigens, triggering cross-reactive immune responses that exacerbate vascular damage and chronic inflammation. The association between *S. mutans* and cardiovascular disease promotes the significance of oral health as a part of systemic illness prevention in light of these complex pathways. Reducing dietary sugar intake, getting frequent dental checks, and practicing good oral hygiene can all help lower the likelihood of *S. mutans* colonization. *S. mutans*'s impact in cardiovascular disorders may also be reduced by antimicrobial treatments that target its biofilms and adhesion characteristics. Interdisciplinary methods between dentistry and cardiology will be essential in tackling the systemic implications of oral pathogens as research continues to reveal the complexity of this bacterial bridge.

### 3.2 Prevention and Therapeutic Approaches

Dental caries, or tooth decay, is mostly caused by *Streptococcus mutans* (*S. mutans*), which has also been linked to cardiovascular conditions such as infective endocarditis. Minimizing the effects of *S. mutans* on heart and dental health requires an understanding of preventative and treatment approaches (Nomura et al., 2020). The process of maintaining a healthy mouth free from illness and other issues (including bad breath) by consistent brushing (dental hygiene) and inter-tooth cleaning is known as oral hygiene. Maintaining good oral hygiene on a regular basis might help avoid tooth problems and foul breath. The two most prevalent forms of dental disease are gum disease, which includes gingivitis and periodontitis, and tooth decay, which includes cavities and dental caries. Although it is generally advised to brush twice a day, it is optimal to do so after each meal. As crucial as brushing your teeth is interdental cleaning, which is cleaning in between your teeth. Because of its inability to reach between teeth, a toothbrush can only remove roughly half of the plaque from the surface of teeth. There are several equipment's available for cleaning in between teeth, such as interdental brushes, floss, and flossettes; everyone is free to choose the one that they like most.

**DIET:** Eating a healthy, balanced diet is crucial for both your general health and dental hygiene. Your diet should contain a lot of fruits, vegetables, dairy products, and whole grains. Peanuts, cheese, yogurt, and milk are a few foods that are particularly beneficial for dental health. By clearing sugars from the tooth surface, these meals may prevent teeth from plaque (Dhage & Chougule, 2019).

### 3.3 Food Advice for Dental Health

- Make the majority of your food choices low in sugar and non-sticking to teeth.
- Every day, children require two to three wholesome snacks and small meals.
- When consuming sweet beverages or meals, pair them with a meal. 4-Select snacks high in protein.
- Drink a lot of liquids.
- The best options are simple, low-fat milk and water.
- Refrain from drinking fresh fruit juice, sports drinks, soft drinks, and fruit drinks.

- Make dietary choices that increase salivation.
- Fresh fruit.
- Veggies (New Zealand Dental Association, 2008).

### 3.4 Virulent Features of Streptococcus Mutans

The ability of *S. mutans* to produce large amounts of organic acids through the metabolism of carbohydrates, its ability to survive at lower pH levels, and its greater capacity to synthesize extracellular glucan homopolymers using sucrose were the three most significant virulent traits for infections. These traits all carried out actions like early adherence, colonization, and biofilm formation on tooth surfaces. It has been known for millennia that sweets lead to dental cavities. Today, however, it is well known that sugar is a preferred food source for cariogenic bacteria such as *mutans streptococci*, which are found in dental plaque. Also, the acid byproducts of metabolic processes are what cause enamel (Daboor et al., 2015).

## 4. CONCLUSION

Tooth decay, or dental caries, is a common oral health problem that affects people of all ages. Despite tooth decay is frequently thought of as a localized issue, recent studies point to a possible connection between it and general wellness, especially cardiovascular health. In this connection, *Streptococcus mutans*, a major cause of dental caries, has been implicated. The purpose of this review is to look into how *S. mutans* contributes the relationship between tooth decay and heart health may be influenced by *S. mutans*, an important factor to the development of dental caries. A key player in the relationship between oral and cardiovascular health is *Streptococcus mutans*, the main bacterium that causes tooth decay. Poor oral hygiene has been linked to an overabundance of *S. mutans*, which raises the risk of systemic infections, including endocarditis, in addition to tooth caries, according to studies. Through irritated gums or dental operations, this bacterium can enter the bloodstream, possibly causing inflammation and assisting in the creation of arterial plaque, which is associated with heart disease. The information now available indicates that maintaining proper oral hygiene may have advantages beyond the oral health benefits,

while additional analysis is required to completely understand the mechanisms involved. Understanding this connection underlines the need of maintaining proper oral hygiene and scheduling regular visits to the dentist to lower the risk of cardiovascular issues and tooth decay. The necessity for multidisciplinary cooperation between cardiology and dentistry is further supported by the possibility that improved preventative and treatment options may result from more investigation into the processes by which *S. mutans* impacts heart health. Individuals may be able to reduce their risk of cardiovascular disease and tooth decay by lowering the load of *S. mutans* and other oral bacteria. A severe condition, dental caries is characterized by the demineralization of tooth structure carried on by acids generated by oral bacteria, mainly *S. mutans*. These microorganisms flourish in dental plaque, a complicated biofilm that builds up on the surfaces of teeth. *S. mutans*'s carcinogenicity is attributed to a number of virulence characteristics, including as its capacity to generate acids, synthesis extracellular polysaccharides for biofilm development, and endure in acidic conditions. There has been plenty of study on the connection between cardiovascular disease and dental health. Several studies have shown a link between an elevated risk of cardiovascular events, including heart attacks and strokes, and periodontal disease, an inflammatory disorder that affects the tissues that support teeth. There is minimal evidence to support a direct connection between dental caries and heart disease, however *S. mutans* has been suggested as a possible mediator. Maintaining proper dental hygiene habits is crucial, as evidenced by the possible connection between *S. mutans*, tooth decay, and heart disease. The burden of *S. mutans* in the oral cavity can be lessened with regular brushing, flossing, and professional dental cleanings. This may also reduce the chance of systemic problems. Additionally, poor dental health may put those with pre-existing cardiac issues at higher risk for infective endocarditis, an infection of the heart valves. Thus, it is essential that these people practice good oral hygiene and, if necessary, seek dental care as soon as possible. The Gram-positive facultative anaerobe *Streptococcus mutans* (*S. mutans*) is a major cause of dental caries and has been linked increasingly to cardiovascular diseases (CVDs). It develops in the oral cavity by creating strong biofilms and sticking to tooth surfaces with extracellular polysaccharides

produced by glucosyltransferases (Gtfs). By digesting dietary carbohydrates into lactic acid, these biofilms provide an acidic microenvironment that demineralizes enamel and Collagen-binding protein (CBP), that allows adhesion to fibrinogen, fibronectin, and type I/IV collagen present in injured vascular tissues and heart valves, is one of *S. mutans*'s primary virulence factors in cardiovascular pathology. Due to the formation of vegetative biofilms by *S. mutans* on heart valves, which can cause inflammation and even valve damage, this characteristic is especially linked to infective endocarditis. The bacterium also secretes serine-rich adhesins that improve its ability to adhere to platelets, promoting aggregation and facilitating in the formation of thrombus. This platelet activity makes people more susceptible to myocardial infarction and stroke by raising their risk of embolism and arterial blockage. Additionally, by binding to toll-like receptors (TLRs) on monocytes and endothelial cells, *S. mutans* causes systemic inflammation by promoting the production of pro-inflammatory cytokines such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF- $\alpha$ ). By encouraging oxidative stress, leukocyte adhesion, and foam cell production in artery walls, these inflammatory mediators promote in endothelial dysfunction, which leads to atherosclerosis. Furthermore, autoimmune reactions may be exacerbated by molecular mimicry between *S. mutans* antigens and host proteins, which would increase cardiovascular damage. By infecting macrophages and changing their lipid metabolism, *S. mutans* may also accelerate the development of atherosclerotic plaque by causing foam cell production, according to emerging data. Its capacity to withstand oxidative stress and escape host immunity enables it to persist in cardiovascular tissues, exacerbating the development of the disease. The function of *S. mutans* in cardiovascular health, in light of these complex processes, emphasizes the need of dental infection control and oral hygiene maintenance as components of preventive cardiology.

## DISCLAIMER (ARTIFICIAL INTELLIGENCE)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc) and text-to-image generators have been used during writing or editing of this manuscript.

## COMPETING INTERESTS

Authors have declared that no competing interests exist.

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