A Review Article The role of *Streptococcus sanguinis* in Normal Human Health and Patients

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ABSTRACT

Background: The bacteria were named *Streptococcus sanguinis*; these bacteria are typically associated with a wholesome plaque biofilm. As a pioneering commensal colonizer of human tooth surfaces, *S. sanguinis* prevents oral pathogens like *Streptococcus mutans* and *Porphyromonas gingivalis* from colonizing dental biofilms, promoting dental biofilm homeostasis. These two species have been shown to possess several virulence factors crucial for the development of infective endocarditis. Nevertheless, it remains unknown how commensal bacteria can occasionally become harmful. The initial colonizer, *S. sanguinis*, is an oral *Streptococcus species* frequently isolated in high abundance as a component of the health-associated microbiome. *S. sanguinis* can bind to the hydroxyapatite in tooth surfaces and start the production of biofilms in the oral cavity by attaching itself to salivary components such as salivary α-amylase. It can utilize various glucose sources for survival. Oral streptococci strains of *S. sanguinis* are less sensitive to C3b deposition than strains of other species. While *S. sanguinis* strain differences in C3b binding significantly affect PMN sensitivity to opsonophagocytic in human peripheral blood. Autoimmune illnesses are a group of heterogeneous conditions characterized by autoreactive immune responses that result in immune system-mediated organ damage. **Conclusion:** Extending the duration of *S. sanguinis*'s early enamel biofilm production appears to enhance demineralization and alter the properties of *S. sanguinis* biofilms during the timeframes investigated in this study and when sucrose is present. When repeatedly exposed to sucrose, S. sanguinis, developing as a monospecies biofilm, exhibits a cariogenic potential, albeit less so than S. mutans. This potential is evident mainly in accelerated demineralization. In addition to preventing microorganisms from entering the bloodstream and internal tissues, the complement system is essential for preserving the homeostasis of the human microbiome, which includes the microbial communities found in the oral cavity. It has been observed that *S. sanguinis* isolated from individuals without caries produces more H_2O_2 than the same species isolated from individuals with multiple carious lesions.

Keywords: *Streptococcus sanguinis*, Biofilm, Oral microbiota, Complement system.
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INTRODUCTION

Characteristics of *Streptococcus sanguinis*:

The bacteria named *S. sanguinis* are typically associated with a wholesome plaque biofilm (1). It is a Gram-positive facultative anaerobe that doesn't produce spores. Like other streptococci, *S. sanguinis* splits its cells along a single axis to form chains or pairs of cocci. *S. sanguinis* has often been characterized as non-motile. Gurung *et al*.'s

discovery that retractable type-IV pili in *S. sanguinis* strain 2908 facilitate surface-associated twitching motility has raised doubts about this recently (2).

As a pioneering commensal colonizer of human tooth surfaces, *S. sanguinis* prevents oral pathogens like *Streptococcus mutans* and *Porphyromonas gingivalis* from colonizing dental biofilms, promoting dental biofilm homeostasis (3). Conversely, *S. sanguinis* frequently causes infective endocarditis (IE) in vulnerable hosts and, ultimately, in young immunocompetent adults (4).

This species has a unique ability to cause cardiovascular infections, as evidenced by the frequent detection of atheromatous plaques (5). Despite being oral commensals, they can elude their niche and result in the deadly condition known as infective endocarditis.

***Streptococcus sanguinis* virulence factors:**

S. sanguinis has been shown to possess several virulence factors crucial for developing infective endocarditis. Nevertheless, it remains unknown how commensal bacteria can occasionally become harmful (6). The methods used to identify the etiologic agents of oral streptococci throughout the past century have had a significant impact on our knowledge of these bacteria. This led to the first disregard of the benefits of oral streptococcal colonization. A new picture began to emerge in 2005 (7), with the conclusion of the first comprehensive study of the oral microbiota of inhabitants. The development of high-throughput sequencing techniques and more sensitive analysis approaches has allowed for the demonstration of a distinct microbiome associated with oral health (8).

The important microorganism that causes dental caries and periodontal disease was polymicrobial (9). *S. sanguinis* is an oral *Streptococcus* species frequently isolated in high abundance as a component of the health-associated microbiome (10). Given the strong association between *S. sanguinis* and oral health, this comparative study can serve as a model for investigating the potential interactions between different species within the bacterial community to affect the composition of a benign dental biofilm. In most cases, *S. sanguinis* is categorized as a chain-forming, catalase-negative, non-spore-forming coccus. Although *S. sanguinis* is not beta-hemolytic, it can induce alpha-hemolysis through hydrogen peroxide (H_2O_2), resulting in a green color on blood agar plates. (11). (Figure 1)

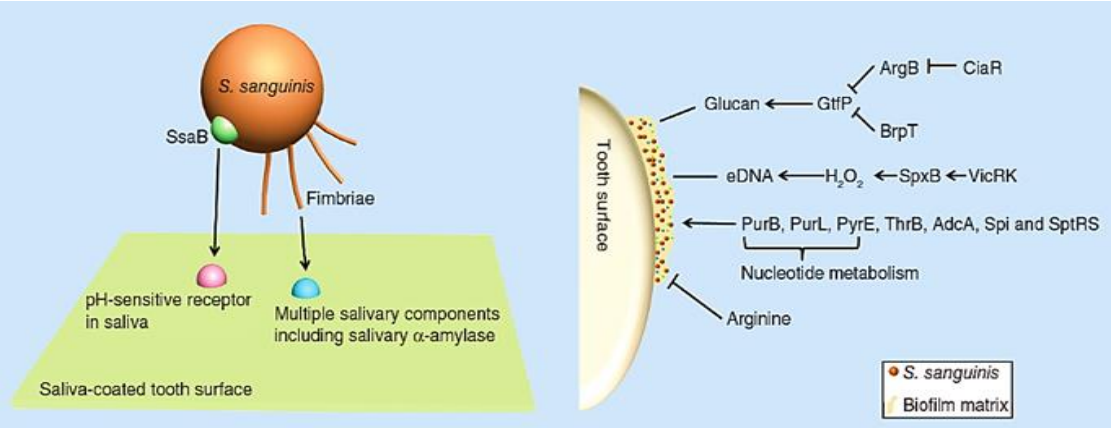

Figure (1): The model shows *S. sanguinis* bacteria identifying salivary pellicle receptors on tooth surfaces and establishing connexons. They also identified various attachment receptor types, such as long-range and pH-sensitive receptors. ArgB expression can be inhibited by CiaR, leading to increased gtfP expression. BrpT deletions can upregulate gtfP, causing biofilm production. (1).

Sporadic cases of infection with *S. sanguinis* were recorded in post-ureteral surgeries. The bacteria cause urinary tract infection (UTI) primarily and sepsis secondary (12). This bacterium is considered one of the most common causes of infective endocarditis, particularly in those with immune-compromised patients or those suffering from cardiac dysfunctions (13).

S. sanguinis and dental caries' relationship:

Streptococcus mitis, *S. sanguinis*, and other members of the viridians group of streptococci are the most common causes of infection after dental operations. While coagulase-negative organisms, such as Enterococci and *Staphylococcus aureus*, were less common and caused infection after dentistry treatments (14).

S. sanguinis is less likely to cause endocarditis and other valvular diseases than other bacteria, such as *Staph. aureus*, after dental surgery (15).

The development of an infectious _S. sanguinis_ biofilm in endocarditis:

In a recent study by (16), it was found that *S. sanguinis* not only causes infective endocarditis and heart valve or endocardial lining disease, but it is also a prominent colonizer in the oral cavity (17). It was found to be a resident of the oral cavity; *S. sanguinis* was known to cause endocarditis and was referred to as "Streptococcus Subacute Bacterial Endocarditis." for "subacute bacterial endocarditis" (18). Along with two other species of Gram-positive cocci, staphylococci and enterococci, oral streptococci, including *S. sanguinis*, were found to be among the top three causes of endocarditis.

Factors influencing _S. sanguinis_ biofilm formation:

Many studies referred to the fact that adults and children who have a proportion of *S. sanguinis* in their saliva and biofilms extracted were free of dental caries, in contrast to those with dental caries. This indicates an important potential protective function for health (19). Clinical studies show that *S. sanguinis* colonizes toddlers' oral cavities before *S. mutans* and before the eruption of their first teeth. Nonetheless, studies conducted in situ have demonstrated that *Streptococcus species* during the initial 4–8 hours of biofilm formation (20). (Figure 2)

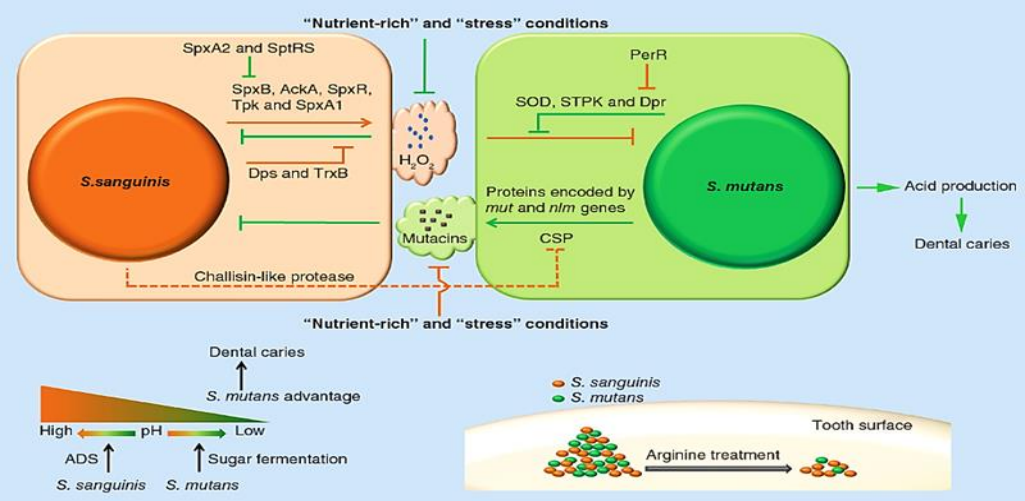

Figure (2): The antagonistic mechanisms of *S. sanguinis* and *S. mutans*. *S. sanguinis* produces H_2O_2, preventing growth, whereas *S. mutans* produces mutacins to stop *S. sanguinis*, *S. mutans* induces dental caries by producing acids from fermentable carbohydrates, whereas *S. sanguinis* preserves pH homeostasis. L-arginine treatment reduces *S. mutans* biomass (1).

S. sanguinis is a very significant species because it is one of the most common causes of infective endocarditis (IE), about 18 to 30 % of cases were caused by *S. sanguinis* (21). Bacteria can enter the circulation through dentistry and poor oral health routine, oral hygiene treatments, including brushing and mastication that may promote a situation of sporadic, temporary bacteremia. Because of inflammation and more serious damage to the oral epithelium, *S. sanguinis* adheres to circulating platelets and attaches to submucosal proteins, such as collagen, after endothelial disruption at locations of injury. These are crucial early steps in the formation of IE (22).

Recognizing the virulence of *S. sanguinis* is lifelong, given the possibility of endocarditis and the inability to treat chronic infections with long-term antibiotics. Currently, enhanced *S. sanguinis* virulence initiation elements are being built toward the creation of treatments specifically designed to eliminate the causes of bacterial endocarditis. (23).

***Streptococcus sanguinis* and the immune system:**

There is evidence that both innate and adaptive immune systems can tolerate *S. sanguinis*, in contrast to other oral streptococci (24). Despite *S. sanguinis's* advantageous role in the oral cavity, it is frequently linked to opportunistic cardiovascular infections in vulnerable hosts (25). Because this association is related to the tolerance and suitability of this type in human blood, in addition to its resistance to Polymorphonuclear leukocytes (PMN) death and cell invasion (26). On the other hand, nothing is known about the functional diversity of different strains of *S. sanguinis*. Preliminary analyses of the 20-25 *S. sanguinis* genomes available in the public domain have shown that the strains have genes that function to produce the nuclear apparatus and cause disease (27).

Pathogenesis of *S. sanguinis* in the oral cavity:

By binding to salivary components like salivary α-amylase, *S. sanguinis* can initiate the formation of biofilms in the oral cavity by binding to the hydroxyapatite present on tooth surfaces. It can utilize various glucose sources for survival (28). When *S. sanguinis* enters the bloodstream systemically, it can act as an opportunistic pathogen. Moreover, if it manages to colonize a damaged heart valve, it could result in infectious endocarditis (29). *S. sanguinis* is associated with bacteremia brought on by dental therapy, regular brushing, and persistent dental lesions. It was first isolated from a patient suffering from subacute bacterial endocarditis (30).

Therefore, epidemiological information may be clinically significant in demonstrating the potential for *S. sanguinis* to survive longer in blood (29). *S. sanguinis* is mainly made up of salivary glycoproteins and microbiological components. It attaches to the film's pellicle molecules and becomes adsorbed on tooth surfaces. The process of adhesion starts when *S. sanguinis's* surface clings to pellicle constituents through hydrophobic and electrostatic interactions (31). Following this, receptors interact with pellicle ligands, including proline-rich proteins and α-amylase/secretory IgA (SIgA) complexes (32).

Complement system with *S. sanguinis*:

Target bacteria use the three known pathways: lectin (LP), alternative (AP), and classical (CP) to activate the complement system. These processes all result in the cleavage of C3-by-C3 convertases. This protein is widely distributed in blood and host tissues, where it is converted into the effector parts C3b and C3a (anaphylatoxin), despite differences in the early stages of microbial identification. Bacteria attaching to erythrocytes, platelets, and other host cells expressing C3b receptors use the highly reactive molecule C3b as a ligand. It also forms covalent bonds with surrounding target microorganisms. C3b is an important opsonin for phagocytes (33).

Functions that elude and undermine complement functions are commonly expressed by microbial pathogens (34). Oral streptococci strains of *S. sanguinis* are less sensitive to C3b deposition than strains of other species (35). While *S. sanguinis* strain differences in C3b binding significantly affect PMN sensitivity to opsonophagocytic in human peripheral blood (36). The complement system is essential for preventing bacteria from entering the bloodstream and internal tissues, and for maintaining the balance of the human microbiome, which comprises the microbial populations in the mouth cavity. After entering the bloodstream through the mouth, *S. sanguinis* needs to adjust to blood conditions and stay away from complement-mediated immunity, a sizable subset of innate immunity linked to host tissue defense and blood clearance (34).

Autoimmune diseases and Streptococcal infection:

Autoimmune illnesses are a group of heterogeneous conditions characterized by autoreactive immune responses that result in immune system-mediated organ damage (37). The clinical manifestations and progression of autoimmune diseases vary greatly. The kidneys, skin, joints, heart, blood vessels, and central nervous system are just a few of the bodily organs that can be impacted by autoimmune illnesses (38). Microbes and autoimmune diseases have been linked in numerous reports (39).(Figure 3).

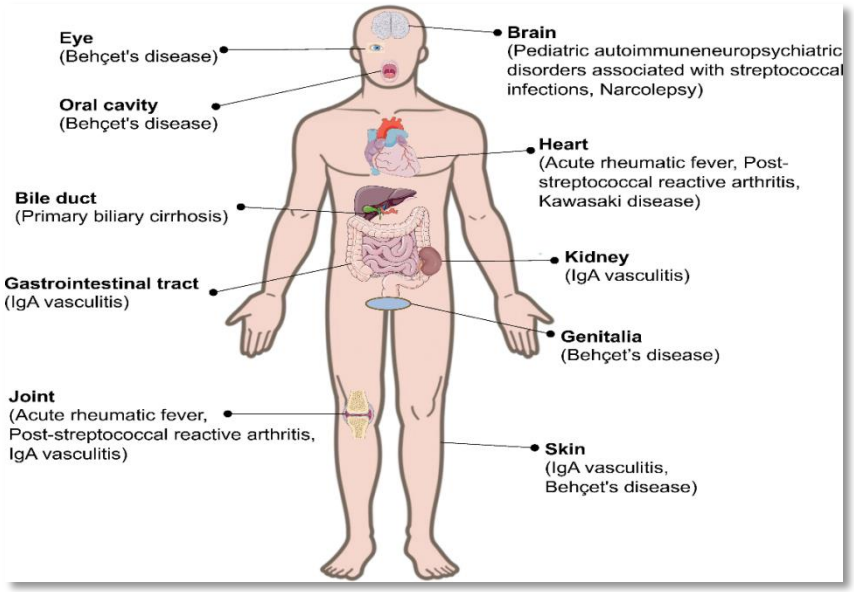

Figure (3): infection with streptococcal bacteria and associated autoimmune disorders. Infections with streptococci can cause autoimmune disorders in many body parts (40).

One of the key components of host defense is leukocytes' capacity to penetrate tissues in response to immunological stimuli (40). On the other hand, inflammation is made worse by overactivating leukocytes, which can result in several autoimmune disorders. In addition to causing a variety of symptoms related to the skin and respiratory tract, Group A *Streptococcus* (GAS) can also occasionally result in autoimmune sequelae that last long after the infection has cleared up because of particular characteristics like molecular mimicry, streptococcal antigens (Sags), direct deposition of streptococcal antigens, xenobiotic-modified bacterial antigens, or organ tropism (Table 1).

Although thoroughly characterized, the precise mechanism by which streptococci cause chronic autoimmunity remains unknown. It is still unclear which frequent streptococcal proteins trigger autoimmunity and how precisely these antigens stimulate immune cell assault in particular organs. The persistent interplay between these two elements and the dearth of appropriate animal models make establishing the cause-and-effect relationship between germs and diseases incredibly challenging. Further study in human and animal models is needed to confirm the importance of streptococci in autoimmune disorders and to identify novel therapeutic and preventive strategies (41).

Table (1): Summarized research on the relationship between autoimmune disorders and streptococcal infection.

Disease	Antigens (Human/Streptococcus)	Immune cells	Mechanism
Acute rheumatic fever	cardiac myosin, tropomyosin, keratin, laminin, vimentin	T cells, B cells	molecular mimicry
IgA vasculitis	NAPlr β2-glycoprotein I	Neutrophils T cells, B cells	deposition of the bacterial antigen molecular mimicry
Kawasaki disease	SPE-A, SPE-G, SPE-J	T cells	superantigens
PANDAS	dopamine D1 and D2 receptors lysogangliosid, tubulin	T cells, B cells	molecular mimicry
Narcolepsy	hypocretin-secreting neurons?	T cells, B cells?	molecular mimicry?
Primary biliary cirrhosis	mitochondrial antigens, histone	T cells, B cells	molecular mimicry xenobiotics
Behçet's disease	human heat-shock proteins	T cells, B cells	molecular mimicry

NAPlr, nephritis-associated plasmin receptor; PANDAS, Pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections.

CONCLUSION

Extending the duration of *S. sanguinis*'s early enamel biofilm production appears to enhance demineralization and alter the properties of *S. sanguinis* biofilms during the time frames investigated in this study, particularly when sucrose is present. When repeatedly exposed to sucrose, *S. sanguinis* developing as monospecies biofilms exhibits a cariogenic potential, albeit less so than *S. mutans.* This potential is mostly evident in accelerated demineralization. In addition to preventing microorganisms from entering the bloodstream and internal tissues, the complement system is essential for preserving the homoeostasis of the human microbiome, which includes the microbial communities found in the oral cavity.

It has been observed that *S. sanguinis* isolated from individuals without caries produces more H2O2 than the same species isolated from individuals with multiple carious lesions.

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دور البكتريا العقدية الدموية في صحة الإنسان الطبيعي والمرضى

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الخلاصة

خلفية البحث : تم تسمية هذه البكتيريا باسم العقدية الدموية (*S. sanguis*)، وعادةً ما ترتبط بطبقة حيوية من البلاك باعتبارها مستعمرًا رائدًا لأسطح الأسنان البشرية، تمنع بكتريا *S. sanguinis* مسببات الأمراض عن طريق الفم مثل *S. mutans* و*Porphyromonas gingivalis* من استعمار الأغشية الحيوية للأسنان، مما يعزز توازن الأغشية الحيوية للأسنان. لقد ثبت أن هذين النوعين يمتلكان العديد من عوامل الضراوة الحاسمة في الإصابة بالتهاب الشغاف المعدي. ومع ذلك، لا يزال من غير المعروف كيف يمكن للبكتيريا المتعايشة أن تصبح ضارة في بعض الأحيان. المستعمر الأولي، *Streptococcus sanguinis*، هو نوع من المكورات العقدية عن طريق الفم يتم عزله بشكل متكرر بكثرة كعنصر من عناصر الميكروبيوم المرتبط بالصحة. يمكن أن ترتبط هذه البكتريا بالهيدروكسيباتيت في أسطح الأسنان وتبدأ في إنتاج الأغشية الحيوية في تجويف الفم عن طريق ربط نفسها بمكونات اللعاب مثل ألفا أميليز اللعابي. يمكنه الاستفادة من مصادر الجلوكوز المختلفة للبقاء على قيد الحياة. تعتبر سلالات المكورات العقدية الفموية من *S. sanguinis* أقل حساسية لترسب C3b من سلالات الأنواع الأخرى، في حين أن اختلافات سلالة *S. sanguinis* في ارتباط C3b تؤثر بشكل كبير على حساسية PMN للخلايا البلعمية في الدم المحيطي البشري. أمراض المناعة الذاتية هي مجموعة من الحالات غير المتجانسة التي تتميز باستجابات مناعية ذاتية التفاعل تؤدي إلى تلف الأعضاء عن طريق الجهاز المناعي.**الهدف من البحث**: تركز هذه المقالة على بكتريا العقدية الدموية ودورها في صحه الانسان ودورها في العديد من الامراض .**الاستنتاج:** يبدو أن تمديد مدة إنتاج الأغشية الحيوية للمينا المبكرة لـ *S. sanguinis* يعزز عملية إزالة المعادن ويغير خصائص الأغشية الحيوية لـ *S. sanguinis* خلال الأطر الزمنية التي تم بحثها في هذه الدراسة وعند وجود السكروز. عند تعرضها بشكل متكرر للسكروز، فإن S. الدموي الذي يتطور على شكل أغشية حيوية أحادية النوع يظهر قدرة على التسرطن، وإن كان أقل من ذلك من *S. mutans*. تتجلى هذه الإمكانية في الغالب في عملية نزع المعادن المتسارعة. بالإضافة إلى منع الكائنات الحية الدقيقة من دخول مجرى الدم والأنسجة الداخلية، يعد النظام التكميلي ضروريًا للحفاظ على توازن الميكروبيوم البشري، والذي يتضمن المجتمعات الميكروبية الموجودة في تجويف الفم. وقد لوحظ أن *S. sanguinis* المعزولة من الأفراد الذين لا يعانون من تسوس تنتج كمية أكبر من H_2O_2 من نفس النوع المعزول من الأفراد الذين يعانون من آفات تسوس متعددة.

الكلمات المفتاحية: العقدية الدموية، الأغشية الحيوية، الميكروبات الفموية، النظام المكمل.