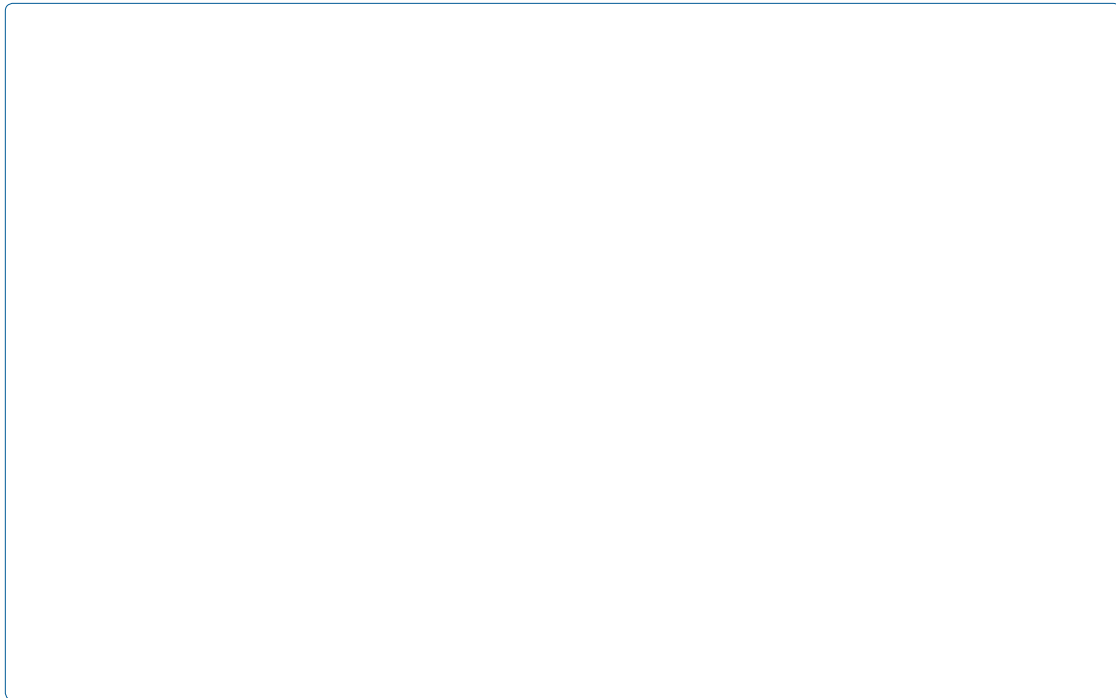


Streptococcus mutans and Dental Caries An In Depth Guide

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Introduction

Dental caries, more commonly known as tooth decay or cavities, is one of the most prevalent chronic diseases globally, affecting individuals of all ages [1]. It is characterized by the demineralization of tooth enamel and dentin, leading to structural damage and, in severe cases, tooth loss [2]. A primary bacterial agent involved in the development of dental caries is Streptococcus mutans (*S. mutans*), a Gram-positive, facultative anaerobe belonging to the lactic acid bacteria group [3]. This bacterium plays a significant role in dental plaque formation, a sticky bio film that adheres to the tooth surface, ultimately promoting cavity formation [4]. Streptococcus mutans (*S. mutans*) is a type of Gram-positive bacterium that plays a key role in the development of dental caries, commonly known as tooth decay or cavities [5]. First identified in the early 20th century, *S. mutans* is known for its ability to thrive in the oral cavity and adhere to tooth surfaces, where it ferments sugars to produce acids [6]. This acid production gradually erodes the tooth enamel, leading to cavities if left unchecked. Due to its significant role in dental health, *S. mutans* is one of the most extensively studied bacteria in the oral microbiome [7].

Characteristics of streptococcus mutans

S. mutans is classified as a Gram-positive coccus, meaning it has a spherical shape and a thick peptidoglycan layer in its cell wall, which retains the crystal violet stain used in Gram staining. It belongs to the

genus Streptococcus, known for comprising bacteria that are generally harmless or beneficial but can also include pathogenic strains. Within the species, several serotypes exist, with serotype c being the most common in the human oral cavity.

This bacterium thrives in the moist, nutrient-rich environment of the mouth, particularly in the dental bio film on tooth surfaces. *S. mutans* is anaerobic, which means it can grow in low-oxygen environments. It can utilize both oxygen-dependent and -independent metabolic pathways, allowing it to persist in various conditions within the oral cavity [8]. Its optimal growth occurs at a pH of around 6.0–7.0, though it can survive in more acidic environments, allowing it to continue thriving even as it produces acid from sugar fermentation. A major factor in *S. mutans*' role in dental caries is its ability to adhere to teeth and form bio films, commonly known as dental plaque. This adherence is facilitated by the production of sticky, extracellular polysaccharides, which allow the bacterium to anchor to the tooth enamel and to other microorganisms.

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oral health outcomes worldwide.

2. Baldwin CL, Parent M (2002) Fundamentals of host immune response against *Brucella abortus*: what the mouse model has revealed about control of infection. *Veterinary Microbiology* 90: 367-382.
3. Ko J, Splitter GA (2003) Molecular host-pathogen interaction in brucellosis: current understanding and future approaches to vaccine development for mice and humans. *Clinical Microbiology Reviews* 16: 65-78.
4. Yagupsky P, Peled N, Press J, Abu-Rashid M, Abramson O (1997) Rapid detection of *Brucella melitensis* from blood cultures by a commercial system. *Eur J Clin Microbiol Infect Dis* 16: 605-607.
5. Shasha B, Lang R, Rubinstein E (1992) Therapy of experimental murine brucellosis with streptomycin, cotrimoxazole, ciprofloxacin, ofloxacin, pefloxacin, doxycycline, and rifampin. *Antimicrobial Agents and Chemotherapy* 36: 973-976.
6. Prior S, Gander B, Irache J M, Gamazo C (2005) Gentamicin loaded microspheres for treatment of experimental *Brucella abortus* infection in mice. *Journal of Antimicrobial Chemotherapy* 55: 1032-1036.
7. Izadjoo MJ, Mense MG, Bhattacharjee AK, Hadfeld TL, Crawford RM, et al. (2008) A study on the use of male animal models for developing a live vaccine for brucellosis. *Transboundary and Emerging Diseases* 55: 145-151.
8. Shemesh AA, Yagupsky P (2011) Limitations of the standard agglutination test for detecting patients with *Brucella melitensis* bacteremia. *Vector Borne Zoonotic Dis* 11: 1599-1601.
9. McFarlane PA, Bayoumi AM (2004) Acceptance and rejection: cost-effectiveness and the working nephrologist. *Kidney International* 66: 1735-1741.
10. Okosun KO, Rachid O, Marcus N (2013) optimal control strategies and cost-effectiveness analysis of a malaria model. *Bio Systems* 111: 83-101.