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THE ROLE OF STREPTOCOCCUS MUTANS IN THE DEVELOPMENT OF EARLY CARIES IN CHILDREN: MECHANISMS OF PATHOGENESIS, MICROBIAL INTERACTIONS AND CLINICAL SIGNIFICANCE

Erkinov Mirsaid Farhod o'g'li,

Ibrohimov Abdurahmon Ibrohim o'g'li,
Safaeva Rukhshona Abdukamalovna
KIMYO International University in Tashkent Tashkent,Uzbekistan

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Resume

Early childhood caries (ECC) is an aggressive form of carious lesion in primary teeth, the etiology of which is traditionally viewed through the prism of an infectious nature. Streptococcus mutans is recognized as the key etiologic agent of this disease. This review summarizes current data on the role of S. mutans in the pathogenesis of ECC, focusing on the mechanisms of primary colonization, the enzymatic activity of glucosyltransferases, the formation of the exopolysaccharide matrix of the biofilm, and synergistic interspecies interactions with Candida albicans, which significantly enhance the cariogenic potential of the consortium. The clinical significance of the timing of infection and threshold concentrations of the pathogen for risk prediction is separately considered. Understanding the molecular basis of S. mutans pathogenicity opens up prospects for the development of targeted preventive strategies that go beyond traditional dietary interventions.

1. Introduction

Early childhood caries (ECC) is defined as the presence of one or more carious lesions (non-cavitated or cavitated) on the surfaces of primary teeth removed due to caries or filled in children under 71 months of age. Unlike caries in permanent teeth, ECC is characterized by a rapid progression and a predilection for the smooth surfaces of the upper incisors, which is related to the peculiarities of microbial colonization in the context of an immature immune system and specific dietary habits.

Historically, caries has been conceptualized as a polymicrobial infection; however, data accumulated over recent decades has firmly established Streptococcus mutans as the central pathogen associated with disease initiation. The aim of this study is to systematize data on the molecular and environmental mechanisms by which S. mutans exerts its cariogenic potential in childhood and to assess the contribution of interspecies interactions to the pathogenesis of ECC.

2. Etiological role of S. mutans: epidemiological evidence

Epidemiological studies convincingly demonstrate an association between S. mutans colonization and the development of ECC. In a classic longitudinal study by Alaluusua et al., children in whom

S. mutans was detected in dental plaque as early as 2 years of age had significantly higher caries intensity scores by 4 years (dmfs = 10.6 ± 5.3) compared with children who acquired the bacteria later (dmfs = 3.4 ± 1.8; $p < 0.005$), and even more so compared with children who remained uninfected (dmfs = 0.3 ± 1.1; $p < 0.0003$). These data served as the basis for the concept of a "window of infectivity" – a period of increased susceptibility to S. mutans colonization occurring approximately between 19 and 31 months of age.

Current research confirms these observations. In a study of children with severe early caries (S-ECC), S. mutans was detected in 100% of them, compared with only 37.5% in the caries-free group ($p = 0.002$). Mean salivary levels of the bacteria in children with S-ECC (6.0 log10 CFU/mL) were significantly higher than those in caries-free children (2.2 log10 CFU/mL; $p < 0.001$). Moreover, salivary mutans streptococci concentrations in the first year of observation correlated more strongly with caries prevalence in the following year ($r = 0.465$) than with those in the year of initial assessment ($r = 0.368$), indicating the prognostic value of microbiological screening.

It's important to note that colonization by S. mutans is not the only sufficient condition: the disease develops only when the infectious agent is combined with a cariogenic diet. However, the persistence and accumulation of S. mutans to levels exceeding 30% of the total culturable plaque microflora is considered a critical threshold, beyond which enamel demineralization becomes irreversible.

3. Molecular mechanisms of cariogenicity of S. mutans

3.1. Glucosyltransferases and the exopolysaccharide matrix

The central component of S. mutans pathogenicity is the production of glucosyltransferases (GtfB, GtfC, GtfD)—extracellular enzymes that catalyze the breakdown of sucrose to form glucans. These polysaccharides form the basis of the exopolysaccharide matrix (EPS) of the biofilm, ensuring bacterial adhesion to the tooth surface and intercellular coaggregation. GtfB predominantly synthesizes insoluble glucans, which, on the one hand, bind tightly to enamel hydroxyapatite and, on the other, create a structural framework that limits acid diffusion and protects microorganisms from innate immune factors and antimicrobial agents.

Regulation of gtf gene expression is controlled by the three-component VicRKX system. Mutations in the vicK gene lead to altered EPS production and disruption of biofilm formation, confirming the key role of this signaling system in the development of the cariogenic phenotype.

3.2. Acidogenicity and acid resistance

*S. mutans*Unique among oral streptococci, S. mutans possesses the ability to actively glycolyze, producing lactic acid, while also exhibiting pronounced acid resistance, allowing it to maintain metabolic activity at pH levels below 4.5. Recent studies have identified an additional mechanism of adaptation: early acid exposure induces S. mutans cell elongation, which is accompanied by enhanced biofilm formation and increased resistance to stress factors. This morphological plasticity

can be considered an adaptive response that promotes pathogen persistence under conditions of frequent consumption of fermentable carbohydrates.

4. Interspecies interactions: synergy of S. mutans and Candida albicans

The traditional focus on S. mutans monocultures has shifted in recent years toward the study of consortia. Of particular interest is the interaction of S. mutans with the yeast Candida albicans, which significantly modulates the virulence of both microorganisms.

The key molecular event in this interaction is the binding of GtfB, secreted by S. mutans, to the mannan layer of the C. albicans cell wall (specifically, to the Als1 and Hwp1 proteins). This results in the in situ synthesis of glucans on the fungal cell surface, which, on the one hand, promotes C. albicans adhesion and, on the other, creates additional binding sites for S. mutans. The resulting mixed biofilm is characterized by increased acid production and increased resistance to antimicrobials compared to monospecific biofilms.

At the transcriptome level, cocultivation with C. albicans leads to the activation of genes associated with carbohydrate transport and metabolism (including components of the phosphotransferase system and ABC transporters), as well as glycogen biosynthesis genes, in S. mutans. Proteomic analysis confirms increased activity of glycolytic enzymes, pyruvate decarboxylase, and glucan synthesis pathways under dual biofilm conditions. Thus, C. albicans acts as an "environmental modifier" that enhances the acidogenic and acid-resistant potential of S. mutans, which is directly relevant to the pathogenesis of ECC, as Candida species are frequently found in the oral cavity of children with severe caries.

5. Clinical implications: from microbiology to prevention

5.1 Prognostic significance of early colonization

Longitudinal studies indicate that children colonized with S. mutans before age 2 years are at highest risk for subsequent dental caries. This supports the use of microbiological screening (e.g., saliva testing for S. mutans) as part of routine dental care in young children, especially those with a positive family history. Mother-to-child transmission of S. mutans, confirmed by molecular epidemiological studies, makes the mother (or primary caregiver) a target for preventive interventions aimed at reducing the bacterial load.

5.2. Limitations of traditional prevention and new horizons

Standard approaches to ECC prevention, based on dietary habit modification and parental hygiene education, demonstrate limited effectiveness: caries recurrence within the first year after surgical treatment reaches 40%. This indicates the need to address the infectious component of the disease. Promising approaches include:

- **Topical antimicrobial therapy**: the use of agents that selectively suppress S. mutans or disrupt the formation of the EPS matrix;
- **Probiotic strategies**: the use of Lactobacillus strains and other microorganisms capable of competing with S. mutans for adhesion sites and modulating biofilm composition;
- **Targeting interspecies interactions**: Inhibition of GtfB binding to the C. albicans cell wall may disrupt the formation of a highly virulent consortium.

6. Conclusion

*Streptococcus mutans*S. mutans remains the central etiologic agent of early childhood caries. Its pathogenicity is realized through a complex of interconnected mechanisms: sucrose-dependent synthesis of exopolysaccharides, which ensures the adhesion and structural integrity of the biofilm; intense acidogenesis combined with pronounced acid resistance; and the ability to enter into synergistic interactions with other representatives of the oral microbiome, in particular with Candida albicans. Epidemiological data confirm that the timing of primary colonization by S. mutans and its quantitative level are significant predictors of caries risk. Further deepening the understanding of the molecular mechanisms of S. mutans pathogenicity and its interspecies interactions creates a scientific basis for the development of new, etiologic-oriented strategies for the prevention and treatment of ECC that can overcome the limitations of existing approaches.

Bibliography

1. Causes, treatment and prevention of early childhood caries: a microbiological perspective. Europe PMC. 2003.
2. Unveiling the complexity of early childhood caries: Candida albicans and Streptococcus mutans cooperative strategies in carbohydrate metabolism and virulence. Journal of Oral Microbiology. 2024.
3. Caries prevalence and salivary mutans streptococci in 0-2-year-old children of Japan. Community Dent Oral Epidemiol. 1991.
4. Multifaceted roles of Candida albicans and Streptococcus mutans in contributing to polybiofilm infections in early childhood caries. Europe PMC. 2025.
5. Streptococcus mutans and Streptococcus sanguinis Colonization Correlated with Caries Experience in Children. Caries Research. 2008.
6. Acid shock triggers virulence surge accompanied by bacterial elongation in Streptococcus mutans. Scientific Reports. 2026.
7. Streptococcus mutans establishment and dental caries experience in children from 2 to 4 years old. Scand J Dent Res. 1983.
8. Dental caries and its relationship to bacterial infection, hypoplasia, diet, and oral hygiene in 6- to 36-month-old children. Community Dent Oral Epidemiol. 2000