



Mutans Streptococci: Acquisition and Transmission

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Abstract

Dental caries is an infectious and transmissible disease. The mutans streptococci (MS) are infectious agents most strongly associated with dental caries. Earlier studies demonstrated that infants acquire MS from their mothers and only after the eruption of primary teeth. More recent studies indicate that MS can colonize the mouths of predentate infants and that horizontal as well as vertical transmission does occur. The purpose of this paper was to demonstrate that these findings will likely facilitate the development of strategies to prevent or delay infant infection by these microbes, thereby reducing the prevalence of dental caries. (Pediatr Dent 2006;28:106-109)

KEYWORDS: MUTANS STREPTOCOCCI, ACQUISITION, TRANSMISSION

Acquisition

The mouth of a normal predentate infant contains only mucosal surfaces exposed to salivary fluid flow. Mutans streptococci (MS) could persist in such an environment by forming adherent colonies on mucosal surfaces or by living free in saliva by proliferation and multiplying at a rate that exceeds the washout rate caused by salivary fluid flow. The oral flora averages only 2 to 4 divisions per day¹ and swallowing occurs every few minutes. Because of this, it is reasonable to assume that bacteria cannot maintain themselves free in saliva by proliferation, but instead must become attached to an oral surface.

Previous studies, reviewed by Gibbons and Van Houte,² have demonstrated that MS have a feeble capacity to become attached to epithelial surfaces. Therefore, it seemed unlikely that these organisms could colonize the mouth of a normal infant before the teeth erupt. Earlier clinical studies reported that MS could not be detected in the mouths of normal predentate infants,³⁻¹⁰ but could after the insertion of acrylic cleft palate obturators or eruption of primary teeth.

A longitudinal investigation by Carlsson and coworkers³ reported that MS were detected in 5 of 25 (20%) infants 12 months to 16 months old. In addition, these organisms were not detected in any of the 25 subjects during their first year of life. Although Carlsson and colleagues did not report the stage of dental development, the age range of 12 to 16 months is compatible with an infant having 6 to

10 primary teeth. Berkowitz and coworkers⁴ reported that MS were detected in 9 of 40 (22 %) infants who had only primary incisor teeth. In addition, these organisms were not detected in 91 normal predentate infants, but were detected in 2 of 10 infants with acrylic cleft palate obturators. In a subsequent study, Berkowitz and colleagues⁵ reported that these organisms were not detected in 16 predentate infants, but were detected in 3 of 43 (7%) infants (mean age=8.9 months) with 1 to 5 primary incisor teeth and in 12 of 42 (29%) infants (mean age=13.8 months) with 6 to 8 primary incisors. Likewise, Stiles and coworkers⁶ could not detect MS in 43 normal predentate children, but isolated these organisms in 12 of 56 (22%) of infants (median age=approximately 14 months) with 6 to 8 primary incisors and in 1 of 38 (3%) of infants (median age=approximately 9 months) with 2 to 4 primary incisors.

Catalanotto and colleagues⁷ failed to isolate these organisms from predentate children or children that harbored only primary incisors; MS were isolated only after the eruption of the first primary molars. Caufield and coworkers⁸ reported that 25% of their infant population (N=46) acquired MS by 19 months of age. Extrapolations of Caufield and colleagues' data from a figure depicting the cumulative probability of MS acquisition as a function of age indicated that approximately 5% of their study population acquired these organisms by approximately 9 months of age. Approximately 15% of the subjects were colonized by approximately 12 months of age. Karn⁹ and coworkers assessed MS prevalence in a population of 149 inner city infants who were enrolled in a WIC Program. Evidence of MS was seen as early as 10 months of age. For children 12 months old, 25% had detectable MS levels; in the 15-month-old age group, 60% were colonized.

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Japanese investigators¹⁰ studied acquisition of MS in a population of 39 children in a Hiroshima day nursery. They reported that the cumulative probability of detecting MS was 20% in children harboring 10 teeth and 40% in children harboring 12 teeth. Collectively, these observations indicated that MS are not detected in the mouths of predentate infants and infrequently colonized the oral cavity until the later stages of primary incisor emergence (approximately 10 to 12 months old). Accordingly, the concept that MS required a nonshedding oral surface for persistent oral colonization became a basic tenet of oral microbial ecology.

More recent clinical investigations¹¹⁻¹³ have demonstrated that MS can colonize in the mouths of predentate infants. Tanner and coworkers¹¹ have demonstrated that the furrows of the tongue appear to be an important ecological niche. Utilizing DNA probe technology, they reported that MS were present in 55% of plaque samples and in 70% of tongue scraping samples of 57 children aged 6 to 18 months living in Saipan. Wan and colleagues^{12,13} reported that MS can be routinely detected prior to tooth emergence. In their study of 172 predentate 6-month-old infants, they found that 50% of the preterm and 60% of the full-term children harbored MS in mouth swab samples. The authors concluded that the detection of these organisms in repeated samples was indicative of colonization. The recent studies¹¹⁻¹³ on acquisition of MS raise doubts that a nonshedding oral surface is required for colonization.

Early acquisition of mutans streptococci and dental caries

Early acquisition of MS is a major risk factor for early childhood caries¹⁴ and future caries experience.¹⁵⁻¹⁹ Alaluusua and Renken¹⁵ performed longitudinal assessment for MS and dental caries in children aged 2 to 4 years. Children who harbored MS in their plaque at age 2 had the most caries by age 4. The mean decayed, missing, and filled primary tooth surface (dmfs) scores in these children was 10.6, whereas in children in whom colonization occurred later the mean dmfs score was 3.4 at age 4 ($P<.005$). Similar observations were made by Kohler and colleagues,¹⁶ who reported that 89% of children with MS colonization by 2 years of age had experienced dental caries by 4 years of age with a mean dfs score of 5.0.

In contrast, 25% of children not infected with MS prior to 2 years of age had experienced dental caries by 4 years of age (mean dfs score=0.3). In another longitudinal study,¹⁷ Grønderfjord and coworkers evaluated 786 children at 1 year of age for caries risk factors that included MS infection, fluoride exposure, dietary habits, and oral hygiene. The children were re-examined at 3½ years of age for the presence of dental caries. The presence of MS at 1 year of age was the most effective predictor of caries at 3.5 years of age. These observations, coupled with the findings of Fujiwara et al¹⁸ and Roeters et al,¹⁹ clearly illustrate that early infection with MS is a significant risk factor for future development of dental caries.

Vertical transmission

Vertical transmission is the transmission of microbes from caregiver to child. The major reservoir from which infants acquire MS is their mothers. The early evidence for this concept comes from bacteriocin typing studies²⁰⁻²² where MS isolated from mothers and their infants demonstrated identical bacteriocin typing patterns. More advanced technology that utilized chromosomal DNA patterns or identical plasmids provided more compelling evidence to substantiate the concept of vertical transmission.²³⁻²⁷

Successful infant colonization of maternally transmitted MS cells may be related to several factors which, in part, include magnitude of the inoculum,²⁸ frequency of small dose inoculations,²⁹ and a minimum infective dose.³⁰ A study conducted by Berkowitz and coworkers²⁸ reported that, when mothers harbored greater than 10^5 colony forming units (cfu) of MS per mL of saliva, the frequency of infant infection was 58%. When mothers harbored 10^3 cfu of MS per mL of saliva or more, however, the frequency of infant infection was 9 times less (6%). These data clearly demonstrate that mothers with dense salivary reservoirs of MS are at high risk for infecting their infants early in life. Accordingly, Kohler and colleagues³¹ demonstrated that a reduction of the salivary level of MS in highly infected mothers can inhibit or delay the establishment of these organisms in their infants. Only 3 of 28 babies (11%) whose mothers were in the experimental group and had their MS reservoirs suppressed by treatment of active caries lesions and topical chlorhexidine gels were infected by the age of 23 months. In contrast, 17 of 38 babies (45%) in the control group whose mothers' levels of MS were not suppressed, were infected by 23 months of age.

A recent study by Li and colleagues³² reported that neonatal factors may also affect risk for early acquisition of MS via vertical transmission. Infants delivered by caesarian section acquired MS 11.7 months earlier than did vaginally delivered infants ($P=.038$). This observation indicates that medical history assessment of the pediatric dental patient should include mode of delivery to identify infants at potential risk for early MS infection.

Horizontal transmission

Horizontal transmission is the transmission of microbes between members of a group (eg, family members of a similar age or students in a classroom). More recent reports³³⁻³⁵ indicate that vertical transmission is not the only vector by which MS are perpetuated in human populations. Horizontal transmission also occurs. This is important given the socioeconomic changes in Western culture over the past 2 to 3 decades. For example, the use of a daycare center or a nanny may provide another vector for acquisition of these organisms. Mattos-Graner and coworkers³³ isolated *Streptococcus mutans* strains from cohorts of Brazilian nursery school children (12 to 30 months of age) and genotyped them by arbitrarily primed polymerase chain reaction and restriction fragment length polymorphism analysis. Of 24 children with 2 to 5 *S. mutans* isolates, 29% carried 2 or

more matching genotypes. The presence of matching genotypes of *S* mutans among children attending one nursery school strongly suggests horizontal transmission.

Van Loeveren and colleagues³⁴ reported data which bacteriocin typed isolates of MS obtained from children 5 years of age and their parents. The results showed that, even when a child acquires MS after the age of 5, there may be similarity between MS in mother, father, and child—indicating that horizontal transmission can occur between family members. Emauelson and Wang³⁵ genotyped isolates of MS from 18 Chinese families consisting of a mother, father, and 3-year-old child without any other siblings. A key observation in their study was that identical genotypes were found between some fathers and their first-born child. The homology of genotypes between spouses was another important observation. These findings also support the concept that horizontal transmission may occur among family members.

Clinical significance

Knowledge regarding the natural history of an infectious disease facilitates a more comprehensive approach toward its prevention (eg, HIV and yellow fever). Studies by Kohler and colleagues,^{16,31} discussed earlier, utilized this concept to inhibit and/or reduce dental caries in young preschool Swedish children by reducing the risk of vertical transmission of MS via suppression of oral MS reservoirs in their highly infected mothers. More recently, Söderling and colleagues³⁶ demonstrated that habitual xylitol consumption (via chewing gum) by mothers was associated with a statistically significant reduction of the probability of vertical transmission of MS at 2 years of age. In a follow-up study,³⁷ Söderling's group reported that, at the age of 5, the dmfs in the children whose mothers habitually used xylitol was reduced by about 70% compared to children whose mothers were in the chlorhexidine and fluoride varnish groups. Clearly, efforts made to reduce the risk for early vertical transmission of MS translates into a decreased risk for dental caries.

Given current knowledge regarding the acquisition and transmission of MS, what can pediatric dentists apply to current clinical practice that will decrease caries risk for the pediatric population? The Dental Bureau of the New York State Department of Health, Albany, NY, has convened an expert panel of pediatricians, obstetricians, periodontists, pediatric dentists, and public health dentists to develop guidelines for oral health care during pregnancy and early childhood. These guidelines are expected to be published in late 2005 as a New York State Health Department publication. The currently drafted recommendations for dental providers addressing the issue of interfering with early acquisition of MS are as follows:

1. Reduce the MS reservoir in the mother, sibling(s), and all of the infant's caretaker(s) by eliminating active dental caries lesions and using agents such as fluorides and chlorhexidine.

2. Alter saliva-sharing activities, such as tasting food before feeding and sharing toothbrushes.
3. Twice daily tooth-brushing of the dentate infant with an appropriate amount of an American Dental Association-approved fluoridated toothpaste.
4. Avoid decay-promoting feeding behaviors.
5. Oral health evaluation of the infant by a dental professional should occur before the first birthday.

In summary, dental caries is an infectious and transmissible disease. Detailed knowledge regarding the acquisition and transmission of infectious agents facilitates a more comprehensive approach toward prevention. MS are important organisms in the initiation and pathogenesis of dental caries. Recent evidence demonstrates that these bacteria are found in the mouths of predentate infants and are acquired via vertical and horizontal transmission from human reservoirs. This information should facilitate the focusing of clinical interventions that prevent or delay infant infection, thereby reducing the prevalence of dental caries.

Conclusions

Based on this paper's findings, the following conclusions can be made:

1. Primary oral infection by mutans streptococci (MS) may occur in predentate infants.
2. Infants may acquire MS via vertical and horizontal transmission.
3. Improvements in the prevention of dental caries may likely be realized through intervention strategies that focus on the natural history of this infectious disease.

References

1. Gibbons RJ, Bacteriology of dental caries. *J Dent Res* 1964;43:1021-1028.
2. Gibbons RJ, Van Houte H, Dental caries. Bacterial adherence in oral microbial ecology. *Annu Rev Microbiol* 1975;29:19-44.
3. Carlsson J, Grahnen J, Jonsson G. Lactobacilli and streptococci in the mouths of children. *Caries Res* 1975;9:333-339.
4. Berkowitz RJ, Jordan HV, White, G. 1975. The early establishment of *Streptococcus mutans* in the mouths of infants. *Arch Oral Biol* 1975;20:171-174.
5. Berkowitz RJ, Turner J, Green P. Primary human infection with *Streptococcus mutans*. *Arch. Oral Biol* 1980;25:221-224.
6. Stiles HM, Meyers R, et al. Occurrence of *Streptococcus mutans* and *Streptococcus sanguis* in the oral cavity and feces of young children. In: Stiles HM, Loesche WJ, O'Brien TC, eds. *Microbial Aspects of Dental Caries*. Vol. 1. London, England: Information Retrieval; 1976:187-199.
7. Catalanotto FA, Shklar II, Keene HJ. Prevalence and localization of *Streptococcus mutans* in infants and children. *J Am Dent Assoc* 1975;91:606-609.

8. Caufield PW, Cutter GR, Sasanayake AP. Initial acquisition of mutans streptococci by infants evidence for a discrete window of infectivity. *J Dent Res* 1993; 72:37-45.
9. Karn T, O'Sullivan D, Tinanoff N. Colonization of mutans streptococci in 8- to 15-month-old children. *J Pub Health Dent* 1998;58:248-249.
10. Tedjogasonko V, Kozai K. Initial acquisition and transmission of mutans streptococci in children at day nursery. *J Dent Child* 2002;69:284-288.
11. Tanner ACR, et al. The microbiota of young children from tooth and tongue samples. *J Dent Res* 2002;81:53-57.
12. Wan AKL, Seow K, et al. Association of Streptococcus mutans infection and oral developmental nodules in prenatate infants. *J Dent Res* 2001;80:1945-1948.
13. Wan AKL, Seow K et al. Oral colonization of Streptococcus mutans in six-month-old prenatate infants. *J Dent Res* 2001;80:2060-2065.
14. Berkowitz RJ. Causes, treatment and prevention of early childhood caries: A microbiologic perspective. *J Can Dent Assoc* 2003;69:304-309.
15. Alaluusua S, Renkonen OV. Streptococcus mutans establishment and dental caries experience in children from 2 to 4 years old. *Scand J Dent Res* 1982; 91:453-457.
16. Kohler B, et al. The earlier the colonization by mutans streptococci, the higher the caries prevalence at 4 years of age. *Oral Microbiol Immunol* 1988;3:14-17.
17. Grindorfjord M, et al. Stepwise prediction of dental caries in children up to 3.5 years of age. *Caries Res* 1995;30:356-366.
18. Fujiwara T, et al. Caries prevalence and salivary mutans streptococci in 0.2-year-old children of Japan. *Community Dent Oral Epidemiol* 1991;19:151-154.
19. Roeters RJM, et al. Lactobacilli, mutans streptococci, and dental caries: A longitudinal study in 2-year-old children up to the age of 5 years. *Caries Res* 1995;29:272-279.
20. Berkowitz RJ, Jordan HV. Similarity of bacteriocins of Streptococcus mutans from mother and infant. *Arch Oral Biol* 1975;20:725-730.
21. Davey AL, Rogers AH. Multiple types of the bacterium Streptococcus mutans in the human mouth and their intra-family transmission. *Arch Oral Biol* 1984;90:453-460.
22. Berkowitz RJ, Jones P. Mouth-to-mouth transmission of the bacterium Streptococcus mutans between mother and child. *Arch Oral Biol* 1985;30:377-379.
23. Caufield PW, Wannemuehler Y, Hensen J. Familial clustering of the Streptococcus mutans cryptic plasmid strain in a dental clinic population. *Infect Immun* 1982;38:785-787.
24. Caufield PW, Childers NK, et al. Plasmids in Streptococcus mutans: Usefulness as epidemiological markers and association with mutacins. In: Hamada S, Michalek S, et al, eds. *Proceedings of an International Conference on Cellular, Molecular, and Clinical Aspects of Streptococcus Mutans*. Birmingham, Ala: Elsevier Science Publishers; 1985:217-223.
25. Caufield PW, Childers NK, et al. Distinct bacteriocins correlate with different groups of Streptococcus mutans plasmids. *Infect Immun* 1985;48:51-56.
26. Caufield PW, Ratanpridakul K, et al. Plasmid-containing strains of Streptococcus mutans cluster within family and racial cohorts: Implication in natural transmission. *Infect Immun* 1988;56:3216-3220.
27. Kulkarni GV, Chan KH, Sandham HJ. An investigation into the use of restriction endonuclease: Analysis in the study of transmission of mutans streptococci. *J Dent Res* 1989;68:1155-1161.
28. Berkowitz RJ, Turner J, Green P. Maternal salivary levels of Streptococcus mutans: The primary oral infection in infants. *Arch Oral Biol* 1981;26:147-149.
29. Loesche W. Role of Streptococcus mutans in human dental decay. *Microbiol Rev* 1986;50:353-380.
30. Van Houte J, Green DB. Relationship between the concentration of bacteria in saliva and colonization of teeth in humans. *Infect Immun* 1974;9:624-630.
31. Kohler B, et al. Preventive measures in mothers influence the establishment of Streptococcus mutans in their infants. *Arch Oral Biol* 1983;28:225-231.
32. Li Y, Caufield PW, Dasanayake HW, et al. Mode of delivery and other maternal factors influence the acquisition of Streptococcus mutans in infants. *J Dent Res* 2005;84:806-811.
33. Mattos-Graner R, Li Y, et al. Genotypic diversity of mutans streptococci in Brazilian nursery children suggests horizontal transmission. *J Clin Microbiol* 2001;39:13-18.
34. Van Loveren C Buijs JF, ten Cate JM. Similarity of bacteriocin activity profiles of mutans streptococci within the family when the children acquire strains after the age of 5. *Caries Res* 2000;34:481-485.
35. Emanuelsson I, Wang X. Demonstration of identical strains of mutans streptococci within Chinese families by genotyping. *Eur J Oral Sci* 1998;106:788-794.
36. Söderling E, Isokangas P, et al. Influence of maternal xylitol consumption on acquisition of mutans streptococci by infants. *J Dent Res* 2000;79:882-887.
37. Isokangas P, Soderling P et al. Occurrence of dental decay in children after maternal consumption of xylitol chewing gum: A follow-up from 0-5 years of age. *J Dent Res* 2000;79:1885-1889.

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