Causes, Treatment and Prevention of Early Childhood Caries: A Microbiologic Perspective

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Abstract

Early childhood caries (ECC) is a virulent form of dental caries that can destroy the primary dentition of toddlers and preschool children. It occurs worldwide, afflicting predominantly disadvantaged children. High-risk North American populations include Hispanic and Native American children, as well as children enrolled in Head Start, a federally funded program for preschool children living in poverty. The prevalence of EEC among these children ranges from 11% to 72%. ECC is an infectious disease, and Streptococcus mutans *is the most likely causative agent; diet also plays a critical role in the acquisition and clinical expression of this infection. Early acquisition of* S. mutans *is a key event in the natural history of the disease. Acquisition may occur via vertical or horizontal transmission. Primary oral colonization by* S. mutans *coupled with caries-promoting feeding behaviours results in accumulation of these organisms to levels exceeding 30% of the total cultivable plaque flora which in turn leads to rapid demineralization of tooth structure. Treatment of ECC is costly because the cooperative capacity of babies and preschool children usually necessitates the use of general anesthesia. Treatment usually consists of restoration or surgical removal of carious teeth along with recommendations regarding feeding habits. However, this approach has resulted in unacceptable clinical outcomes, and relapse rates of approximately 40% have been reported within the first year after dental surgery. Primary prevention of ECC has largely been restricted to counselling parents about caries-promoting feeding behaviours. This approach has also had minimal success. Newer strategies addressing the infectious component through use of topical antimicrobial therapy appear promising.*

MeSH Key Words: *child, preschool; dental caries/microbiology; streptococcal infections/epidemiology*

© J Can Dent Assoc 2003; 69(5):304–7
This article has been peer reviewed.

Early childhood caries (ECC) is a particularly virulent form of dental caries that is characterized by an overwhelming infectious challenge and is associated with unusual dietary practices. ECC initially presents with smooth-surface carious lesions affecting the primary maxillary incisors (PMIs). As the disease progresses, decay appears on the occlusal surfaces of the primary maxillary first molars, with subsequent spread to other primary teeth, resulting in the eventual destruction of the primary dentition.

ECC is a public health problem that continues to affect babies and preschool children worldwide. A comprehensive review of the epidemiology of ECC showed that its prevalence varies from population to population; however, disadvantaged children, regardless of race, ethnicity or culture, are most vulnerable.[1] High-risk North American populations include many different groups. Almost 30% of 125 Mexican-American children (8–47 months of age) living in the Yakima Valley (Washington State) were reported to have ECC.[2] Serwint and others[3] reported that 20% of 110 Mexican-American children (18–36 months of age) who were patients of a hospital-based pediatric practice in Los Angeles had ECC. Results from epidemiology studies show that Native American children are at high risk for ECC. The prevalence of ECC among Navajo (n = 1,463) and Cherokee (n =144) children enrolled in Head Start, a federally funded program for preschool children living in poverty, was 72% and 55% respectively.[4] Similar observations were reported by Kelly and Bruerd,[5] who observed a high prevalence of ECC among 515 American Indian (41.8%) and Alaskan Native (66.8%) children enrolled in Head Start programs in Oklahoma and Alaska respectively. Albert and others[6] found that the prevalence of ECC in a population of 260 preschool Inuit children was 65%. Non-Native American children enrolled in Head Start programs also display high levels of ECC. In an examination of 1,230 children (3–5 years of age) in 37 Head Start

programs in Arkansas, Louisiana, New Mexico, Oklahoma and Texas, the prevalence of ECC was 18.5% for 3-year-olds, 22.4% for 4-year-olds and 27.9% for 5-year-olds.[7] The prevalence of ECC in Head Start programs in 2 Ohio communities (total of 200 children 3.5–5 years of age)[8] and St. Thomas, U.S. Virgin Islands (375 children 3–5 years of age)[9] was 11% and 12% respectively. Overall, then, the prevalence of ECC for these high-risk North American populations ranged from 11% to 72%.

Because ECC is an infectious disease, this paper reviews current information regarding the causes, treatment and prevention of ECC from a microbiologic perspective.

Causes

Microbiological Risk Factors

Microbial Characteristics of ECC: Bacteriologic studies[10–12] have demonstrated that in children with ECC, *Streptococcus mutans* regularly exceeded 30% of the cultivable plaque flora. This dense level of dental infection was associated with carious lesions, white spot lesions and sound tooth surfaces near the lesions. Conversely, *S. mutans* typically constitutes less than 0.1% of the plaque flora in children with negligible to no caries activity.[13] These observations, together with other published results,[14–16] clearly illustrate the concept that ECC is an infectious disease and that *S. mutans* is the most likely infectious agent; clearly diet also plays a critical role in the clinical expression of this infection.

Early colonization by Streptococcus mutans is a major risk factor for ECC and future dental caries.

Early Acquisition of S. mutans: The mouth of a normal predentate infant contains only mucosal surfaces exposed to salivary fluid flow. *S. mutans* could persist in such an environment by forming adherent colonies on mucosal surfaces or by living free in the saliva and duplicating at a rate exceeding the washout rate of salivary flow. Because the oral flora averages only 2 to 4 divisions per day[17] and swallowing occurs every few minutes, it is reasonable to assume that bacteria cannot maintain themselves in saliva by proliferation, but instead must become attached to an oral surface. Previous studies (reviewed by Gibbons and Van Houte[18]) demonstrated that *S. mutans* has 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 eruption of teeth. Earlier clinical studies[19–24] reported that *S. mutans* could not be detected in the mouths of normal predentate infants; instead, the organisms were found only after the insertion of acrylic cleft palate obturators or eruption of primary teeth. Accordingly, the concept that *S. mutans* required a nonshedding oral surface for persistent oral colonization became a basic tenet of oral microbial ecology. However, more recent clinical studies[25–27] have demonstrated that *S. mutans* can colonize the mouths of predentate infants; the furrows of the tongue appear to be an important ecological niche. Tanner and others,[28] using DNA probe technology, reported that *S. mutans* was present in 55% of plaque samples and 70% of tongue scraping samples of 57 children 6–18 months of age living in Saipan, Commonwealth of the Northern Mariana Islands, western Pacific. These recent studies on acquisition of *S. mutans* raise doubt that a nonshedding oral surface is required for colonization.

Early Acquisition of S. mutans *and Dental Caries:* Early colonization by *S. mutans* is a major risk factor for ECC as well as future dental caries. Alaluusua and Renkonen[29] performed longitudinal assessment for *S. mutans* colonization and dental caries in children 2–4 years of age; children who harboured *S. mutans* in their plaque at the age of 2 had the most caries activity by the age of 4. The mean DMFS score in these children was 10.6, whereas in children in whom colonization occurred later, the mean DMFS score was 3.4 at age 4 ($p < 0.005$). Similar observations were made by Kohler and others,[30] who reported that 89% of children with *S. mutans* colonization by 2 years of age had carious lesions by 4 years of age and a mean DMFS score at that time of 5.0; in contrast, only 25% of children not infected with *S. mutans* before 2 years of age had experienced dental caries by 4 years of age, and they had a mean DMFS score of 0.3. In another longitudinal evaluation, Gindefjord and others[31] evaluated 786 children at 1 year of age for caries risk factors (*S. mutans* infection, exposure to fluoride, dietary habits, oral hygiene) and re-examined them at 3.5 years of age for the presence of dental caries. The presence of *S. mutans* at 1 year of age was the most effective predictor of caries at 3.5 years of age. These observations, together with other published results,[32,33] illustrate that early infection with *S. mutans* is a significant risk factor for future development of dental caries.

Transmission of S. mutans*:* The major reservoir from which infants acquire *S. mutans* is their mothers. The evidence for this concept comes from several clinical studies in which *S. mutans* strains isolated from mothers and their babies exhibited similar or identical bacteriocin profiles[34–36] and identical plasmid or chromosomal DNA patterns.[37–41] Successful colonization of infants by maternally transmitted *S. mutans* cells may be related to several factors, including magnitude of the inoculum,[42] frequency of small-dose inoculations[13] and minimum infective dose.[43] Berkowitz and others[42] reported that the

frequency of infant infection was approximately 9 times greater when maternal salivary levels of the organism exceeded 10^5 colony-forming units (CFU)/mL than when maternal salivary reservoirs were less than or equal to 10^3 CFU/mL (58% vs. 6%). Suppression of maternal reservoirs of *S. mutans* prevented or delayed infant infection,[44] and only 3 (11%) of 28 babies whose mothers underwent suppression of the *S. mutans* reservoir (by dental treatment and topical antimicrobial therapy) were infected by 23 months of age; in contrast, 17 (45%) of 38 babies in the control group (whose mothers did not undergo *S. mutans* suppression) were infected. In both groups the percentage of infected babies increased with age; nevertheless, at 4 years of age fewer babies in the test group were infected.

Two recent reports indicate that vertical transmission is not the only vector by which *S. mutans* is perpetuated in human populations. Mattos-Graner and others[45] isolated *S. mutans* from groups of nursery school children (12–30 months of age) and genotyped the isolates with primed polymerase chain reaction and restriction fragment-length polymorphism analysis. They reported that many children had identical genotypes of *S. mutans*, which indicated that horizontal transmission may be another vector for acquisition of these organisms. In addition, van Loveren and others,[46] using bacteriocin typing, demonstrated that when a child acquires *S. mutans* after the age of 5 years, there may be similarity in *S. mutans* strains in mother, father and child, which indicates that horizontal transmission can also occur among family members. These findings are of importance given the socio-economic changes in Western culture over the past 2 decades (for example, the use of daycare facilities for babies and preschool children when both parents are employed).

Dietary Risk Factors

Although ECC is an infectious disease, the role of diet in acquisition of the infection[47] and development of the disease[48] is critical. Children with ECC have frequent and prolonged consumption of sugars from liquids.[2,49–52] Caries-promoting sugars such as sucrose, glucose and fructose, contained in fruit juices and many infant formula preparations,[53–55] are readily metabolized by *S. mutans* and lactobacilli to organic acids that demineralize enamel and dentin. The use of nursing bottles and "sippy cups" enhances the frequency of exposure. This type of feeding behaviour during sleep intensifies the risk of caries, as oral clearance and salivary flow rate are decreased during sleep. In addition, caries-promoting feeding behaviours result in an increase in the magnitude of dental reservoirs of *S. mutans*.[48]

Clinical Significance

The preceding narrative strongly suggests that the first event in the natural history of the infectious disease ECC is primary infection by *S. mutans*. The second event is accumulation of *S. mutans* to pathogenic levels, secondary to frequent and prolonged dietary exposure to caries-promoting sugars. The third event is rapid demineralization of enamel, which results in cavitation of tooth structure.

Treatment

The current standard of care for treatment of ECC usually necessitates general anesthesia, with all of its potential complications, because the level of cooperative behaviour of babies and preschool children is less than ideal. In a 1993 study, Milnes and others[56] found that the costs associated with the treatment of ECC for a Canadian aboriginal population (for travel, lodging, medical care and facilities, and general anesthesia) were a significant drain on government resources. A 1994 report indicated that the cost of treating a child with ECC exceeded US$2,000.[57] More recent data have shown that costs have escalated. For example, the average cost for facilities and general anesthesia, excluding dental services, at Strong Memorial Hospital, University of Rochester Medical Center, is US$3,500. Thus, this disease places a huge burden on third-party payers (insurance companies and government medical welfare agencies), as well as on parents least likely to be able to afford it.

Treatment of ECC is usually restricted to surgical removal or restoration of carious teeth coupled with recommendations regarding feeding habits. Restorative dentistry has little to no long-term impact on oral *S. mutans* populations[58] and, as discussed below in the Prevention section, recommendations regarding feeding behaviours have had minimal impact. Not surprisingly, the clinical outcomes for treatment of ECC are poor. Sheehy and others,[59] using a telephone survey, found that 23% of children treated for ECC under general anesthesia required restorations or extractions after the initial dental surgery. In another study,[60] 52% of the cohort treated under general anesthesia presented with new smooth-surface enamel lesions within 4–6 months after dental surgery. Eidelman and others,[61] using a retrospective chart review, reported that 57% of the study cohort who had been treated under general anesthesia required treatment for new carious lesions within 6–24 months after the initial dental surgery. In another retrospective study[62] of 42 children with ECC treated under general anesthesia at the Franciscan Children's Hospital and Rehabilitation Center in Boston, 45% had experienced relapse by the end of 12 months after dental surgery. Given the morbidity and cost associated with treatment of relapse (e.g., general anesthesia, sedation, physical restraint), the current standard of care for ECC, involving

treatment under general anesthesia, results in unacceptable clinical outcomes. New treatment strategies (e.g., chemotherapeutic, behavioural) must be developed to address the causative factors associated with relapse if improvements in clinical outcomes are to be realized.

Prevention

Prevention of cariogenic feeding behaviours is one approach to preventing ECC. Regrettably, educating parents about this risk factor has had minimal success. Johnsen[63] reported that 78% of parents of children with ECC had attempted to substitute water for a cariogenic liquid (e.g., apple juice, formula) in the bedtime nursing bottle; this observation strongly suggests that these parents were aware of the feeding practices associated with ECC. In a survey of parents of 169 Inuit children with ECC, 54% of the parents knew that naptime and bedtime bottle feedings may be associated with ECC.[6] Likewise, 25 of 38 Mexican-American parents whose children had ECC were aware of the feeding behaviours associated with ECC.[2] However, given the high prevalence of ECC in certain groups, it appears that information and knowledge do not always translate into appropriate parenting practices. Thus, the parents of children with ECC are frequently aware of the dietary practices associated with the development of the disease, but they may not implement changes in feeding behaviours.

A promising approach toward primary prevention of ECC is to develop strategies that target the infectious component of this disease, for example by preventing or delaying primary acquisition of *S. mutans* at an early age through suppression of maternal reservoirs of the organism.[44]

Another approach is to prevent *S. mutans* from accumulating to pathologic levels through topical application of antimicrobial agents. This approach was recently applied in a group of Puerto Rican babies at high risk for ECC.[64] The study population consisted of 83 subjects who were 12 to 19 months of age at the time of their entry into the study. Inclusion criteria for the study included unremarkable medical history, presence of 4 PMIs with no visible defects, caries-free status (accordingly to clinical criteria), use of a nursing bottle containing a cariogenic substance at naptime or bedtime, and 2 consecutive *S. mutans*-positive cultures from pooled PMI plaque. The subjects were randomly assigned to 2 groups. In the experimental group (n = 39), a 10% povidone–iodine solution was applied to the dentition bimonthly for 1 year; in the control group (n = 44) a placebo solution was applied in the same manner. Treatment failure was defined as the appearance of one or more white spot lesions on any of the PMIs during the study period. Using the Kaplan–Meier procedure, the authors estimated that 91% ± 5% of experimental subjects and 54% ± 9% of control subjects experienced 12 months of disease-free survival (log-rank test, 2-sided p = 0.001). Therefore, bimonthly topical application of a 10% povidone–iodine solution to the dentition of babies at high risk for ECC increased disease-free survival. It is important to determine, through larger and more in-depth clinical trials, if this effect remains after the antimicrobial agent is withdrawn before introducing 10% povidone–iodine therapy into clinical practice as a primary prevention modality for ECC. ◆

Acknowledgment: *This paper is based on a presentation given at the October 2001 CAPD annual meeting. The manuscript was supported in part by NIDCR grant DE12595.*

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The author has no declared financial interests.

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