Early childhood caries: overview and recent findings

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Early childhood caries (ECC) is a relatively new term that describes rampant dental caries in infants and toddlers.[1] In many cases, it is thought to be initiated and exacerbated by inappropriate feeding with a nursing bottle. The condition, when associated with the bottle habit, has been characterized as first affecting the primary maxillary anterior teeth, followed by involvement of the primary molars. Mandibular incisors generally are not affected, reportedly due to the child's tongue in the suckling position protecting these teeth from the cariogenic challenge.[2,3] Fass is credited with first using the term nursing bottle mouth to describe this caries pattern.[4] Terms to describe this condition have evolved during the last two decades to include nursing caries, nursing bottle caries, and baby bottle caries. In 1985, the term baby bottle tooth decay was proposed by the Healthy Mothers-Healthy Babies Coalition as an alternative that would be more appropriate for patient acceptance and would focus attention on the potential damage of using a nursing bottle.[5,6]

Although the combination of infection with mutans streptococci and the caretaker-reported practice of taking a bottle to bed may be a good predictor of who among certain populations will have nursing caries (Table), there have been recent challenges to the conventional wisdom that inappropriate bottle use and high levels of oral infection with mutans streptococci are the sole etiologic factors of this condition. Such concerns have given impetus to replacing the language associating cause and effect with the term early childhood caries, which reflects a less certain understanding of the etiology.[1] While the basic concepts of early infection with mutans streptococci and inappropriate feeding with a cariogenic diet remain important factors in the etiology of ECC, the exclusive focus of a child sleeping with a bottle containing milk or other sugar-containing substances is being explored. This review examines these controversies and recent information regarding the etiology, implications, and prevention of dental caries in infants and toddlers.

TABLE. AN EXAMPLE OF THE ABILITY OF BOTTLE USE AND MUTANS STREPTOCOCCI (MS) INFECTION TO IDENTIFY AND PREDICT CARIES*

		Caries†		
		Yes	*No*	*Total*
Bottle Use & MS‡	*Yes*	30	4	34
	No	7	73	80
	Total	37	77	114
	sensitivity =	81.1%		
	specificity =	94.8%		
	PPV =	88.2%		
	NPV =	91.3%		

* Unpublished data from a longitudinal study to identify caries risk factors in preschool children that was conducted on inner-city Head Start children in Hartford, Connecticut between 1991and 1993. For detailed population description and methods, see reference 42.

† Any form of caries present at third examination.

‡ At baseline examination, 1) parent reported that the child had taken a bottle to bed, and 2) child had mutans streptococci infection > 50 cfu per MSKB plate.

Etiology

It is widely accepted that the group of cariogenic microorganisms, mutans streptococci, is associated with ECC. Children with ECC reportedly have elevated oral levels of mutans streptococci,[7-9] which generally are acquired from their mothers.[10-12] Such high numbers of acidogenic microorganisms combine with frequent carbohydrate intake to produce abundant acid that lowers plaque pH for extended periods and demineralizes the child's teeth. Since most of these microbiological studies were conducted on a limited number of subjects using bacterial samples collected well after the disease process began, the age at which children become infected with mutans streptococci is not well understood. Determining the age of infection is critical to understanding the disease process, the efficacy of microbial screening tests at different ages, and the optimal period during which to intervene with preventive strategies.

One recent report suggested that mutans streptococci are acquired during a window of infectivity between ages 19 and 31 months.[13] However, earlier studies have found colonization of mutans streptococci in the oral cavity at younger ages. One study showed that children as young as 11 months were infected with mutans streptococci;[14] another found that 12 of 42 children at the same developmental stage were infected with mutans streptococci,[10] and

a third found these microorganisms in five of 25 toddlers having six to 10 primary teeth,[15] a tooth developmental stage corresponding to an age range of 12 to 16 months.[16] Moreover, a child as young as 11 months has been reported to have frank carious lesions.[17] Clearly, additional research is required to better understand when children of various caries risk acquire mutans streptococci.

Prolonged and night-time bottle-feeding practices in infants and toddlers generally are thought to provide the carbohydrate source that promotes high acid production by mutans streptococci. Yet evidence suggests that blaming sleeping with a bottle of milk may oversimplify the cause of rampant caries. Several studies have reported that the majority of U.S. preschool populations take, or have taken, a bottle to bed.[18, 19] In one study of U.S. Head Start children, 86% of children with caries of the maxillary anterior incisors were reported to have taken a bottle to bed, but surprisingly, 69% of those who did not have maxillary anterior caries also reportedly took a bottle to bed.[20] In another study, 90% of children in a population with and without caries were bottle-fed between 12 and 18 months of age, yet the prevalence of nursing caries was only 20%.[21] Since this feeding pattern is pervasive, it follows that parents of children with ECC often answer "yes" to the question, "Do you put your child to bed with a bottle?".[20] Thus, it is logical that the bottle-to-bed habit is inferred as the cause of early childhood caries.

In addition to the lack of a clear association between feeding patterns and all cases of early childhood caries, there is controversy regarding the effect of various bottle contents on the carious process. Of course, there is no cariogenic challenge if the bottle contains only water. However, the majority of 6-month- to 5-year-old children are put to bed with a bottle with contents other than water.[18] It is well recognized that liquids in the bottle that contain sucrose are cariogenic, yet the potential cariogenicity of the most common bottle contents—milk and infant formulas—remains unclear. Although not tested in humans, rats fed milk as the sole source of nutrition do not develop caries,[22, 23] and rats given sucrose-milk solutions had fewer caries than those given sucrose-water solutions.[24]

Several reports suggest why milk may be less cariogenic than other sugar-containing liquids. Phosphoproteins in milk have been shown to inhibit enamel dissolution,[25, 26] antibacterial factors in milk interfere with the oral microbial flora,[27] and cariogenic bacteria may not be able to utilize lactose as an energy source as readily as sucrose.[28] Additionally, milk has been shown to remineralize artificially demineralized enamel in vitro.[29] While the cariogenicity of milk per se is unclear, it may be the vehicle for more cariogenic substances. Parents are known to combine milk or milk/formula with other food products or sugar.[26] Worse, nutritional information labels show that sucrose is an ingredient in some infant formulas.

Another controversial yet poorly documented caries risk is the potential cariogenicity of at-will breast feeding. There are case reports associating prolonged or night-time breast feeding and ECC.[30–33] However, one unpublished report surveying more than 1,000 children breast-fed ad libitum for 1 to 4 years found a 5% prevalence of maxillary anterior caries.[34] One cannot dismiss a possible association between reported rampant caries in these cases and dietary practices other than breast feeding. Further study is required to determine the prevalence of ECC in exclusively breast-fed children, and whether other child-rearing practices, such as lack of restriction in eating snacks,[35] could contribute to caries in breast-fed children as well as in bottle-fed children.

Children with caries in the primary maxillary anterior teeth, independent of their ages, generally are regarded as having nursing bottle caries.[2, 3, 36] Although it is likely that the disease is due, at least in part, to a prolonged use of the baby bottle, other causes of caries affecting the anterior teeth cannot be ruled out. Children who are 4 and 5 years old, an age by which bottle use generally has been discontinued, may develop caries in the maxillary anterior teeth. This late involvement of the maxillary anterior teeth is characterized by a higher prevalence of mesial lesions on the central incisors than when ECC is diagnosed at a younger age (Figure). Data from developing countries also suggest that caries on anterior primary teeth cannot, in all cases, be attributed to inappropriate bottle use. For example, in Beijing, China, where the prevalence of caries in maxillary anterior teeth has been reported to be 45% in 4-year-old children,[37] baby bottles generally are not available. Perhaps other etiologies in developing countries, such as linear hypoplasia of primary teeth associated with malnutrition,[38] may contribute to the prevalence of this condition. It is interesting to speculate that visible or subclinical enamel hypoplasia may be a factor for the high caries prevalence found in the primary teeth of U.S. preschoolers from families of low socioeconomic status.

Implications of early childhood caries

The seriousness and societal costs of ECC are enormous, especially among racial or ethnic minorities. The prevalence of caries in 3- to 5-year-old U.S. Head Start children has been reported to range as high as 90%.[39] There is considerable evidence that children who experience ECC continue to be at high risk for new lesions as they get older, both in the primary and permanent dentitions.[40–43] Perhaps the high levels of infection by cariogenic microorganisms, or the establishment of poor nutritional practices, may be determinants of caries progression.[44]

Treatment of ECC is expensive, often requiring extensive restorative treatment and extraction of teeth at an early age. Estimates of the cost of restoring the teeth alone may exceed $1,000 per child.[45] In addition to these

expenses, general anesthesia or deep sedation may be required because such young children lack the ability to cope with the procedures. General anesthesia to facilitate dental treatment adds between $1,000[6] and $6,000[34] to the cost of dental care.

One study also implicates ECC as contributing to other health problems. Children with ECC were shown to weigh less than 80% of their ideal weight, and to be in the lowest 10th percentile for weight.[46] Perhaps the pain or infections associated with ECC may make it difficult for affected children to eat. Alternatively, poor nutritional practices may be responsible for both the reduced body weight and the caries. Thus, the consequences of ECC are a significant problem not only in monetary terms to parents and federal or state agencies paying for the care, but in potential risks to health and discomfort of the child with the disease. The potential association of ECC with growth lags is an important observation that requires additional study.

Prevention of early childhood caries

ECC prevention has focused on educational programs to alter children's feeding practices and to reduce levels of mutans streptococci infection. However, there has been surprisingly little scientific effort to test methods for reducing the prevalence of this disease. An intensive education program using training aids—manuals, counseling booklets, posters, and bumper stickers with messages about preventing baby bottle tooth decay—was employed in 16 Native American communities in an attempt to alter child feeding practices. Surveys of ECC prevalence before and after the intensive educational programs showed that ECC decreased to 43% from 57%.[47]

Another common educational approach to alter ECC is individual parent counseling. A small study was performed with 17 mothers of infants with initial signs of ECC. The self-reports on stopping the use of the bottle, substitution of noncariogenic substrate in the bottle, and use of the fluoride gel showed that the caretakers generally could not, or would not, comply with the preventive regimen. Additionally, a presurvey showed that 12 of the 17 caretakers admitted being aware of the potential cariogenicity of sweet liquids in the bottle.[48]

Strategies to reduce the transmission of cariogenic microorganisms to offspring also have been studied as methods to prevent ECC. In one study, 37 first-time mothers with high levels of mutans streptococci were given a preventive regimen, including use of chlorhexidine, to reduce their levels of mutans streptococci infection. Follow-up data showed that the mutans streptococci level of the 3-year-old children of these mothers was 41%, compared with 70% in the control group. More importantly, the caries prevalence was 16% in the test group versus 43% in the control group.[49,50] In a similar study, 70 mothers with high levels of mutans streptococci were given a semiannual treatment of chlorhexidine and sodium fluoride. After three years, the children of the mothers in the experimental group had a lower colonization of mutans streptococci and lower caries incidence than those in the control groups.[51] In contrast to these favorable findings, a recent report attempted to reduce transmission of mutans streptococci to infants by giving the mothers' dentitions six applications of I_2-NaF at the time of the child's tooth eruption. This study found that mutans streptococci colonization and caries experience of the test group did not differ from controls.[52]

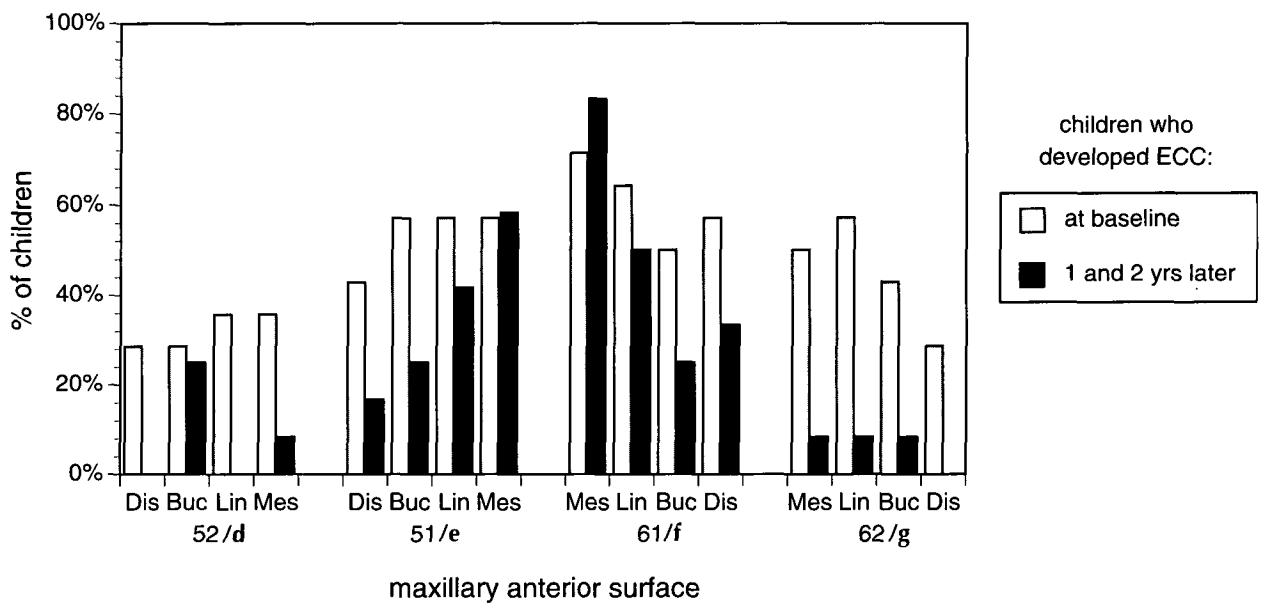

Figure. Patterns of early childhood caries in Head Start children from the inner-city of Hartford, Connecticut. Children (mean baseline age, 3.8 years) were examined for dental caries once annually for 3 years. Baseline refers to children who were determined to have early childhood caries at first examination; 1 and 2 years later refers to children who were determined not to have early childhood caries at first examination but did have it at second or third examination.

Additional methods to foster preventive behaviors in parents whose children are at high risk for ECC need to be explored. Psychological approaches, such as self-efficacy enhancement and performance feedback techniques, may increase parents' confidence in their ability to carry out recommendations and perform oral health preventive behaviors for their children. Self-efficacy enhancement involves raising an individual's confidence to perform certain behaviors. Performance feedback provides observable success in performing a beneficial behavior. Although these techniques have not been applied to behavioral changes required to prevent ECC, considerable literature shows their effects in other health areas.[53]

Besides considering behavioral techniques to change adverse health behaviors, perhaps intense preventive interventions that do not rely on patient compliance also should be examined as methods of reducing ECC. In some groups, lack of preventive behaviors and deeply entrenched feeding practices may be so difficult to change that it would not be practical to alter these behaviors. Results from frequent professional toothbrushing or professional administration of an antimicrobial agent, or fluoride, have not been reported, and may need to be considered to reduce caries incidence in such groups. The focus of such programs is to place the responsibility for caries prevention on the dental health professional, rather than on the parent.

Summary

Early childhood caries is of epidemic proportions in some U.S. minority populations and in developing countries, yet a review of the literature reveals numerous conflicting reports and unanswered questions regarding the etiology and prevention of the disease. Better knowledge of the cause of early childhood caries and effective strategies to reduce its risk should produce enormous reductions in initial and long-term dental treatment costs, as well as the pain and suffering of affected children.

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