Early Childhood Caries (ECC): what's in a name?

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ABSTRACT. Aim It is evident from the number of published scientific papers on Early Childhood Caries (ECC) that interest in this problem has grown in recent years. Many authors have been trying to devise a clear definition or classification for ECC. The aim of this review was to inventory the prevalence of ECC and to seek a consensus regarding definition and diagnosis. Further attention was paid to the aetiological factors including the role of microorganisms. Finally, education, parenting and treatment procedures were discussed. Methods For this review, epidemiological studies on caries prevalence in children aged between 0 and 36 months were compiled through a systematic approach using Medline. Review This clearly showed that ECC continues to be a serious public health problem and that there is a great variety of definitions and diagnoses used worldwide, reflected in the prevalence data. This review confirms the multicausal aetiology and the need for further research. The authors strongly support the recommendations formulated at the workshop in Bethesda 1999, and the policy statements by the AAPD. Conclusion More efforts should be made to reach the high risk groups within populations, in order to reduce the prevalence of ECC and S-ECC (Severe Early Childhood Caries) and consequently to ameliorate the quality of life of these children. Long-term intervention studies are required for the evaluation of these efforts.

KEYWORDS: Early Childhood Caries, Case definition, Prevalence, Diagnosis, Aetiology.

Introduction
For years, rampant caries in the primary dentition of infants and young children has been described by several terms including: nursing bottle caries, nursing caries, rampant caries, baby bottle caries, baby bottle tooth decay, milk bottle syndrome and prolonged nursing habit caries. Supported by the report of the Bethesda workshop [Drury et al., 1999] the term Early Childhood Caries (ECC) is actually preferred.

ECC is a serious oral health problem, especially in disadvantaged communities in both developing and industrialized countries in which undernutrition is common. In many cases, it is thought to be initiated and exacerbated by inappropriate feeding with a nursing bottle. Nowadays, however, more attention is paid to environmental factors, which supports the widely accepted multicausal aspect of dental caries.

The aim of this review was to inventory the prevalence of ECC as reported in the literature and to look for a consensus regarding definition and diagnosis. Further attention was paid to the aetiological factors including the role of microorganisms. Finally, education, parenting and treatment procedures were discussed.

Clinical description
The term Early Childhood Caries (ECC) as the specific virulent form of caries in the primary dentition of infants and young children seems to replace all other terms, as noted above, ever used to indicate a severe form of caries at a very young age (<3 years). These terms, however, are useful as they describe the problem and ascribe a possible cause in a form that could be understood by parents, caretakers and the public. Although the ‘bottle’ has been described as the most frequent cause, there are others that could also lead to ECC. The term ‘nursing caries’ is more inclusive, but it assumes that breastfeeding or other nursing practices alone could cause the condition. Therefore, the term ‘Early Childhood Caries’ has become widely accepted in the most recent literature as a more general term that includes breast-feeding, as well as the use of sweetened pacifiers, which may be significant.
Furthermore there is a strong belief that environmental factors such as social deprivation are most important [Ramos-Gomez et al., 2002; Quinonez et al., 2001].

**Early Childhood Caries (ECC)** is a form of early, moderate and late dental decay that affects the primary teeth of infants and toddlers. It develops in tooth surfaces that are usually at low risk for caries, such as the labial surfaces of maxillary incisors and lingual and buccal surfaces of maxillary and mandibular molars. ECC usually begins with the maxillary primary incisors, initially developing a dull white demineralized band, the so-called ‘white spot’, along the gingival margin. As the condition progresses, caries develops and may evolve to a complete destruction of the crown, leading to root stumps. ECC always begins with an early stage; starting with white or brown spots, appearing at the gum line. In the moderate stage, cavitation starts and caries begins to spread to the maxillary molars. In the severe stage, the caries process destroys the maxillary teeth, and spreads to the mandibular molars. Finally, in very severe cases the mandibular incisors are affected (Table 1). The pattern of caries development is characteristic for the disease and is affected by the sequence of eruption of the primary teeth, the duration of the causative behaviour and the patterns of tongue movement and oral muscular action [Horowitz, 1998].

**Classification**

During the last 20 years, different research groups have attempted to develop classification systems for ECC. In a first approach [Wyne, 1999], the severeness of ECC can be defined in three types and associated with different aetiology. In Type I (mild to moderate) ECC was defined as the existence of ‘isolated carious lesion(s)’ involving incisors and/or molars. The most common causes are usually a combination of semi-solid or solid food and lack of oral hygiene. In Type II (moderate to severe) ECC was described as ‘labiollingual lesions’ affecting maxillary incisors, with or without molar caries, depending on the age of the child and stage of the disease. Typically are the unaffected mandibular incisors. The cause is usually inappropriate use of a feeding bottle or at-will breast-feeding or a combination of both, with or without poor oral hygiene. Finally, a Type III (severe) ECC was described as carious lesions affecting almost all teeth including the mandibular incisors. A combination of cariogenic food substances and poor oral hygiene is the cause of this type of ECC.

A system developed by Johnston and Messer [1994] classifies ECC into 3 main patterns:
- lesions associated with developmental defects (pit and fissure defects and hypoplasia);
- smooth surface lesions (labial-lingual lesions, approximal molar lesions);
- rampant caries.

The latter was defined as having caries in 14 out of 20 primary teeth, including at least one mandibular incisor.

Another, but not specifically ECC, classification system is the Caries Analysis System (CAS) of Ismail and Sohn [1999]. The CAS defines 4 patterns of caries in the primary teeth, according to the site of occurrence. This classification system excludes the buccal and lingual surfaces of the maxillary canines, the buccal, lingual and mesial surfaces of the mandibular canines, and all tooth surfaces of the mandibular incisors. The fissure pattern was 3 to 4 times more likely to develop caries compared with disease-free children. Children with the maxillary anterior pattern had 2.4 times greater two-year increments of pit and fissure caries compared with caries-free children and they also had 8 times more caries increments in the buccal/lingual and proximal areas than caries-free children.
A fourth classification system, proposed by Veerkamp and Weerheijm [1995], claims to account for the stage of development of the dentition and severity of dental caries (initial and cavitated). This classification system assumes that dental caries occurs in successive stages starting late in the first year (10 months) and ending in the fourth year of life (48 months). The four stages were referred to as: initial, damaged, deep lesions and traumatic. At each stage, a different group of teeth are involved and dental caries can range from enamel demineralization (opaque white demineralization) to cavitation involving enamel and dentine.

Despite all valuable efforts to classify ECC lesions clinically, there is also a great need for diagnosing and reporting the condition for research purposes. In this respect, a workshop was held in April, 1999, in Bethesda, USA. In a report from this meeting Drury et al. [1999] (Table 2) defined ECC as “the presence of 1 or more decayed (non-cavitated or cavitated lesions), missing (due to caries), or filled tooth surfaces” in any primary tooth in a child 71 months of age or younger. In children younger than 3 years of age, any sign of smooth surface caries is indicative of Severe Early Childhood Caries (S-ECC). From ages 3 through 5, 1 or more cavitated, missing (due to caries), or filled smooth surfaces in primary maxillary anterior teeth, or a decayed, missing, or filled score >4 (age 3), >5 (age 4), or >6 (age 5) surfaces constitute S-ECC (Table 2). The participants of the workshop, noted above, recommended that the term ‘Severe Early Childhood Caries’ refers to children with ‘atypical’, ‘progressive’, ‘acute’ or ‘rampant’ pattern of dental caries.

### Prevalence of ECC

Epidemiological studies on caries prevalence in children aged between 0 and 36 months were compiled through a systematic search of published literature and was carried out in a series of stages:
- a literature search was performed via the Medline database using a keyword filter including: Early Childhood Caries, nursing caries, nursing bottle caries, baby bottle tooth decay, baby bottle syndrome, baby bottle caries and rampant caries.
- non-English abstracts were not considered.

Existing review articles or papers dealing with nomenclature, definition or diagnosis were especially taken into consideration.

From a systematic review of clinical diagnostic criteria of ECC [Ismail and Sohn, 1999], it became obvious that the prevalence of ECC varied from 2.1% in some welfare centers in Sweden to 85.5% in rural Chinese children. This wide variation was due to the differences in case definition and diagnostic criteria of ECC and to the wide range of areas worldwide, both industrialized as deprived.

The accurate prevalence of ECC in a population is, however, very difficult to determine, as toddlers and preschool age children in general are difficult to examine. Moreover, they are not readily accessible for examination [Horowitz, 1998]. Significant cultural and ethnic differences in feeding practices, the location of dental decay and the number of teeth involved in the process, also explain the difficulty of standardizing and optimizing diagnostic and epidemiological criteria for ECC. According to the
American Association of Pediatric Dentistry (AAPD), the decay level in 3 to 5 year old children may be as high as 90% in some populations [Horowitz, 1998]. It is assured, however, that the national prevalence of ECC in the USA can be estimated between 3 and 6% [Horowitz, 1998], which is consistent with the reported ECC prevalence in western countries (≤5%) [Johnston and Messer, 1994].

Nevertheless, reported prevalences of ECC seem to vary from country to country and from area to area. As the above mentioned review by Ismail and Sohn [1999] summarized all studies until 1998, Table 3 gives an overview of prevalence data found worldwide since that time. As there are also national and local data and as well data from industrialized and developing countries, it is obvious that all these data should be interpreted with caution and should not be used to calculate a mean prevalence of ECC for the world. The only common finding from these studies, is that in all cases the most affected teeth were the maxillary incisors. Non-cavitated carious lesions on smooth surfaces were more prevalent than cavitated lesions in primary teeth in children aged 6 to 18 months.

Once over the age of 18 months, cavitated carious lesions become more apparent [Grindefjord et al., 1993]. One study reported that the highest prevalence of ECC is found in the 3 to 4 year old age group and that boys are significantly more affected compared to girls, aged between 8 months and 7 years [Ramos-Gomez et al., 2002].

### Table 3 - An overview of prevalence data on ECC reported in the literature since 1998.

<table>
<thead>
<tr>
<th>Country/ City/ State</th>
<th>Authors</th>
<th>Age group</th>
<th>Prevalence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>The Netherlands</td>
<td>Weerheijm et al., 1998</td>
<td>28 months</td>
<td>9.3%</td>
</tr>
<tr>
<td>USA</td>
<td>Horowitz, 1998</td>
<td>1-3 years</td>
<td>3-6%</td>
</tr>
<tr>
<td>Puerto Rico</td>
<td>Lopez del Valle et al., 1998</td>
<td>6-47 months</td>
<td>62.6%</td>
</tr>
<tr>
<td>Brazil</td>
<td>Dini et al., 2000</td>
<td>3-4 years</td>
<td>46%</td>
</tr>
<tr>
<td>Rome (Italy)</td>
<td>Petti et al., 2000</td>
<td>3-5 years</td>
<td>7.6%</td>
</tr>
<tr>
<td>Zagreb (Croatia)</td>
<td>Lulic-Dukic et al., 2001</td>
<td>2-5 years</td>
<td>30%</td>
</tr>
<tr>
<td>Brazil</td>
<td>Rosenblatt et al., 2002</td>
<td>12-36 months</td>
<td>28.5%</td>
</tr>
<tr>
<td>Sao Paolo (Brazil)</td>
<td>Santos and Soviero, 2002</td>
<td>0-36 months</td>
<td>41.6%</td>
</tr>
<tr>
<td>Ghent (Belgium)</td>
<td>Martens et al., 2004a</td>
<td>24-36 months</td>
<td>18.3%</td>
</tr>
</tbody>
</table>

### Bacterial relationships of ECC

**Streptococcus mutans.** This organism is known to colonize the oral cavity as soon as hard dental tissue is present. These bacteria are vertically transmitted from mother or caregiver in general to the child. Their virulence expression is strongly associated with consumption of carbohydrates, especially sucrose. The latter explains their abundance in active carious lesions. It should be stressed that controversy exists about the fact that individual carious teeth in the same mouth can harbour a different population of S. mutans strains, or at least the same genotype in considerably different proportions. Consequently, S. mutans is by far the most important microrganism in the caries process. The role of non-mutans streptococci is still unclear, despite their extreme abundance in the oral cavity.

**Other bacteria.** On the other hand, Lactobacilli comprise less than 1% of the total cultivable oral microflora and still their numbers appear to reflect the carbohydrate consumption of the host. They can be found in carious dentine, saliva and on mucosal surfaces. Because Enterococci have only been recovered in very low numbers from several oral sites (in immuno and medically compromised patients), little is known about their role in the caries process. Several Actinomycoses species have been cultured from the mouth, but from the literature it is clear that they should be associated with health, rather than with the initiation of carious lesions. Veillonella species are lactate dependent and as a consequence they are abundantly present in
established carious lesions. Table 4 summarizes all microorganisms associated with the caries process.

**Aetiology**

*Dietary factors.* Many studies suggest that children with ECC have a high frequency of sugar intake, not only from fluids given in a nursing bottle, but also from sweetened solid foods. This dietary characteristic is likely to be one of the most significant caries risk factors in ECC.

Although improper nursing bottle-feeding habits are the most frequently cited causes of ECC, the disease may occur in children who are breastfed at will [Weerheijm et al., 1998], and beyond one year of age, and in those who are given sweetened pacifiers or frequent sugar-containing snacks [Horowitz, 1998]. The cariogenic contents of the nursing bottle include bovine and human milk, nursing formulas, sweetened milk, fruit juices, carbonated and non-carbonated soft drinks. The frequency and the duration of the habit, along with the ingestion of cariogenic solids (e.g. biscuits), are a very important aspect of this condition [Johnston and Messer, 1994; Peressini, 2003]. Ad libitum exposure to cariogenic substances during the night intensifies the risk of caries, as oral clearance and salivary flow rate are decreased during sleep [Milnes, 1996; Berkowitz, 2003].

*Milk.* Milk (human and bovine) and infant milk formulas present a complex role in the aetiology of ECC as they contain protective components against caries such as calcium, phosphorous, casein and other proteins, which provide a protective organic coating on enamel. Bovine milk has a higher concentration of calcium and phosphorous than human milk, bound in organic and inorganic molecules and also in free ionic form. In vitro

### Table 4 - Oral bacterial associations with caries reported in the literature.

<table>
<thead>
<tr>
<th>Bacterial species</th>
<th>References</th>
<th>Typical characteristics</th>
<th>Additional information</th>
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<tbody>
<tr>
<td><strong>Mutans streptococi</strong></td>
<td></td>
<td></td>
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<tr>
<td>S. mutans</td>
<td>Aluluusua et al., 1996&lt;br&gt;Marchant et al., 2001&lt;br&gt;Tanzer et al., 2001</td>
<td>- fermentation of simple carbohydrates&lt;br&gt;- tolerance to low environmental pH levels</td>
<td>- associated with caries activity in young children&lt;br&gt;- vertically transmitted&lt;br&gt;- only within the presence of hard oral tissues (teeth, prosthodontics)</td>
</tr>
<tr>
<td>S. sobrinus</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Non-mutans streptococci</strong></td>
<td>Marchant et al., 2001</td>
<td></td>
<td></td>
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<tr>
<td>S. salivarius</td>
<td></td>
<td>- uncertain role in the caries process&lt;br&gt;- extremely abundant in the oral cavity</td>
<td></td>
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<tr>
<td><strong>Lactobacilli</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>L. fermentum</td>
<td>Marsh and Martin, 1999&lt;br&gt;Marchant et al., 2001&lt;br&gt;Tanzer et al., 2001</td>
<td>- number in saliva reflects simple carbohydrate consumption by the host</td>
<td>- dorsum of the tongue&lt;br&gt;- usually cultured from carious dentine&lt;br&gt;- comprise less than 1% of the total cultivable microflora</td>
</tr>
<tr>
<td>L. casei</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Enterococci</strong></td>
<td>Tanzer et al., 2001</td>
<td>- not frequently found in the human oral cavity</td>
<td>- immuno and medically compromised patients have been associated with their presence in the oral cavity</td>
</tr>
<tr>
<td><strong>Actinomyces</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A. naeslundii</td>
<td>Marchant et al., 2001&lt;br&gt;Tanzer et al., 2001</td>
<td>- carbohydrate users&lt;br&gt;- not associated with initiation of ECC</td>
<td>- isolated from both carious and sound surfaces&lt;br&gt;- more associated with health&lt;br&gt;- the most predominant Actinomyces species in plaque of ECC individuals is A. israelii</td>
</tr>
<tr>
<td>A. odontolyticus</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A. israelii</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Veillonella</strong></td>
<td>Bradshaw and Marsh, 1998&lt;br&gt;Marchant et al., 2001</td>
<td>- lactate users, thus abundant in carious dentine</td>
<td></td>
</tr>
</tbody>
</table>
studies reported enamel to be 20% less soluble in an acidic buffer if initially exposed to bovine milk. Infant milk formulas, bovine and human breast milk contain relatively high concentrations of lactose, which is potentially cariogenic [Yiu and Wei, 1992]. Under conditions conducive to ECC, human milk can be more cariogenic than bovine milk due to its lactose content, 7 versus 4.8% respectively, while the concentrations in infant formulas vary considerably.

Soya-based formulas could be considered as a good substitute for natural milks, but the added carbohydrates used in these formulas are mostly sucrose derivates [Horowitz, 1998]. Under normal dietary conditions, milk is minimally cariogenic and may even be cariostatic [Ripa, 1988]. However, it is important to emphasize that adding sucrose to milk renders milk cariogenic. This does not contradict the clinical evidence attributing ECC to milk, as affected children may have aberrant, rather than normal dietary habits, in association with inadequate oral hygiene and a minimal fluoride exposure.

Fruit juices and soft drinks. These contain a lot of sucrose and have a low pH (pH 3–4), which presents a potentially erosive and cariogenic effect. Loss of enamel from excessive consumption of fruit drinks in children has been documented in several studies. The abusive use of fruit juices and soft drinks in combination with inappropriate remineralization activity by saliva eventually result in a predominant enamel demineralization process [Seow, 1998] and are a common finding in ECC, since they are usually given at will to the child and preferentially in a bottle.

Syrup additives (e.g. vitamin preparations). These may be added by parents to the nursing fluid. Sucrose sweetened medication, such as paediatric antibiotic preparations have also been implicated in the development of ECC [Horowitz, 1998].

Salivary factors. It is known that the salivary flow rate is at the lowest during the night, due to the circadian rhythm. With regard to ECC, the continuous feeding of sugar-containing substances at night time, when the flow rate is the lowest, increases the caries risk of the child significantly. If cariogenic liquids are consumed frequently, teeth may be exposed to cariogenic conditions for lengthy periods with only short intervals allowing salivary remineralization of demineralized enamel [Ripa, 1988].

As the hard dental tissues are immunologically inactive, specific salivary (host) defense mechanisms against bacterial colonization and pathogenic activity of cariogenic organisms are essential. These host immune mechanisms include specific immune factors, derived from saliva (serum immunoglobulin A, sIgA) or serum and gingival crevicular fluid (immunoglobulin G, IgG) and non-specific antimicrobial systems derived mainly from saliva, and phagocytic cells which transude through the gingival crevice [Lehner et al., 1976; Twetman et al., 1981; Tenovuo et al., 1987].

Oral clearance. In children with ECC, the sleep-time consumption of sugar is a common characteristic. The cariogenicity of the substrate increases significantly, as the length of contact time between the plaque and substrate increases. The clearance of glucose is the slowest on the labial surfaces of the maxillary incisors and the buccal surfaces of the mandibular molars. These site differences in oral clearance may explain in part the distribution of the carious lesions in ECC which are characteristically localized to the maxillary primary incisors and first molars [Ripa, 1988; Milnes, 1996].

Oral hygiene. Primary infection with streptococcus mutans by vertical transmission is unavoidable, but measures should be taken to reduce the bacterial numbers that are transferred, for example by avoiding transfer by sharing spoons or cleaning a pacifier in the mothers mouth. Tooth brushing with fluoridated toothpaste by the parents or caretakers should start as soon as the first primary teeth erupt and this at least two times a day. This recommendation however is not appropriate for areas where adequate fluoridation is present (e.g. Ireland), which implies that the use of fluoridated toothpastes in children could be prohibited. The use of a pea size of paste, however, can probably avoid any risk. A gentle sideways scrub using a multi-tufted toothbrush and an adapted toothpaste, low in fluoride concentration (≤500 ppm), are recommended by the European Academy of Paediatric Dentistry [Oulis et al., 2000]. Furthermore, brushing more than once a day and having less than two in-between meals a day is strongly recommended [Martens et al., 2004b].

Environmental and living condition factors. ECC is one of the most prevalent diseases of infants and toddlers from families with a low income and from families with an immigrant and ethnic minority background [Hallett, 2000]. Currently, although ECC is recognized as an infectious disease, its extent and severity vary with cultural, genetic and socioeconomic influences. Significant negative correlations have been shown between the socioeconomic level of a family and the prevalence of ECC in that same family, and between poor oral hygiene and the prevalence of ECC [Santos and
Ethnic minority background, low socioeconomic environments, single parents, and minimal educational background are consistent predictors of dental disease in childhood. However, not all minority children living under these circumstances do consistently experience ECC and the opposite has not been shown to be true either [Quinonez et al., 2001]. Social class may influence caries risk in several ways. Individuals from lower socioeconomic status experience financial, social and material disadvantages that compromise their ability to care for themselves, obtain professional health care services, and live in a healthy environment, all of which can lead to reduced resistance to oral and other diseases. Socioeconomic status is generally measured by indicators of human capital, such as income, education or occupational prestige that offer advantages to individuals and families.

Another approach is to assign a social status position based on ecological measures derived from place of residence [Reisine and Psoter, 2001]. There are, however, relatively few case-control or longitudinal studies that assess the relationship between the socioeconomic status and the incidence of caries among young children. Moreover, it should be considered that an interpretation of socioeconomic status indicators can vary by cultural context and that caution should be exercised in generalizing the findings for other countries. Therefore, cultural factors must be considered in any intervention to reduce or prevent ECC.

### AAPD policy statement on Early Childhood Caries

1. Infants should not be put to sleep with a bottle. Ad libitum nocturnal breast-feeding should be avoided after the first primary tooth begins to erupt.

2. Parents should be encouraged to have infants drink from a cup as they approach their first birthday. Infants should be weaned from the bottle at 12 to 14 months of age.

3. Repetitive consumption of any liquid containing fermentable carbohydrates from a bottle or no-spill training cup should be avoided.

4. Oral hygiene measures should be implemented by the time of eruption of the first primary tooth.

5. An oral health consultation visit within 6 months from eruption of the first tooth and no later than 12 months of age is recommended to educate parents and provide anticipating guidance for prevention of dental disease.

6. An attempt should be made to assess and decrease the mother’s/primary caregiver’s mutans streptococci levels to decrease the transmission of cariogenic bacteria and lessen the infant’s or child’s risk of developing ECC.

### Prevention and parental education

As a result of improper parenting ECC appears to be a problem of over-indulgence and lack of parental restraint rather than of abusive neglect or lack of education [Ripa, 1988; Eronat et al., 1992]. Information on ECC can be distributed to new parents from birthing centers, paediatricians’ offices, maternal clinics and dental offices. Families with infants who are at high risk of developing ECC should be especially targeted. Through prevention-oriented educational programs, prospective parents and new parents can be alerted about the condition and the causes of ECC [Ripa, 1988].

The American Academy of Pediatric Dentistry (AAPD) revised its policy on ECC in 2003. The AAPD discourages inappropriate feeding practices of infants and toddlers and encourages appropriate preventive measures (Table 5). Children should be weaned from the bottle or breast by 12 months and they should not be put to bed with a bottle containing liquids other than water. Weaning methods include progressively diluting the liquid in the nursing bottle until it is completely replaced with water or offering a pacifier that has not been dipped in sweet substances [Johnston and Messer, 1994; Reisine and Psoter, 2001].

### Treatment

Usually, ECC is diagnosed when the prevention and treatment should already have been started. The treatment of ECC is multifactorial and depends on the patient’s and parents’ motivation toward a dental
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Conclusion and recommendations
From the above review it becomes clear that ECC is not only an infectious dental disease, but there are also socioeconomic factors involved. ECC is still a serious public health problem, but with a great variety of definitions and diagnoses used worldwide. For this reason the authors strongly support the recommendations formulated at the workshop in Bethesda, 1999, and support the policy documents by theAAPD.

Unfortunately, most children with ECC are seen by a dental practitioner when the disease has already reached extensive proportions and the children already complain of toothaches and difficulties during eating. Therefore it would be advisable to reach these children in time and to inform the parents about the possible causes of ECC. Parental guidance is the main corner stone of a campaign to prevent ECC. Parents should get the information before the child is born and the first dental visit of the infant and mother should be before the primary teeth erupt at about 8 months of age.

Parents should be instructed about the cariogenic potential of certain foods and drinks and the cariogenic potential of various ‘family-bound habits’. These should include cleaning the child’s pacifier in their own (mother’s) mouth, sharing spoons, no tooth brushing, prolonged breast feeding, nursing bottles with sucrose containing drinks during the night etc.

Finally, more efforts should be made to reach the high risk groups within populations, in order to reduce the prevalence of ECC and S-ECC and consequently to ameliorate the quality of life of these children. Long-term intervention studies are required for evaluation of these efforts.

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